TO

Mr. Brent Truskowski

U.S. EPA Region 6

FROM

Steve Calhoun

ERM-Southwest, Inc.

SUBJECT Arkwood, Inc. Site

DATE September 18, 1990

As you requested, another copy of the MMI Public Comment document is enclosed. If we can be of further assistance, please call.

SIGNED Steve Calhoun

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TECHNICAL SUPPORT FOR PUBLIC COMMENTS OF MASS MERCHANDISERS, INC.

Arkwood, Inc. Site
Omaha, Arkansas

Prepared for:

Mass Merchandisers, Inc.

SEPTEMBER 14, 1990

Prepared By:

ERM-Southwest, Inc.

008652

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September 14, 1990

Ms. Ellen D. Greeney
Community Relations Coordinator
U.S. EPA (6H-MC)
1445 Ross Avenue
Dallas, Texas 75202-2733

HAND DELIVERY

Re: Comments of Mass Merchandisers, Inc. on EPA's Proposed Plan of Action for the Arkwood, Inc. Site, Omaha, Arkansas

Dear Ms. Greeney:

Mass Merchandisers, Inc. submits these comments on EPA's Proposed Plan of Action for the Arkwood site and on the Administrative Record. As set forth more fully below and in the supporting documents, MMI vehemently opposes EPA's proposal for on-site incineration of all affected materials at the Arkwood site. Total on-site incineration of all affected materials is not warranted by site conditions, is not cost effective when compared to other remedial alternatives, and is not acceptable to residents of the local community. The Feasibility Study Report states that consolidating and capping the affected soils, coupled with off-site incineration of the more heavily contaminated materials, (i.e., Alternative D in the Feasibility Study), would comply with ARARs and provide overall protection of human health and the environment. If EPA insists on treatment of the affected soils, which we respectfully submit would be in error, any form of treatment at the site--even including incineration--must be preceded by sieve and wash to reduce volume, increase homogeneity of the materials to be treated, and enhance cost effectiveness.

DISCUSSION

1. Conditions at the Arkwood Site Pose No Significant Risk to Human Health and the Environment.

The conditions at the Arkwood site are fully evaluated in the Remedial Investigation Report, which was approved by EPA. This investigation identified no significant adverse environmental impact from conditions related to the Arkwood site. In addition, an Endangerment Assessment examined the potential for adverse human health effects based upon various hypothetical exposure scenarios. So long as minimal restrictions are placed on use of the Arkwood site for residential use, all of the hypothetical exposure scenarios resulted in an estimated risk prior to remediation that is well within the range ordinarily accepted by the Agency after remediation is complete. EPA approved this Endangerment Assessment, but later recalculated the estimates of risk to human health using newly adopted Toxicity Equivalency Factors (TEF) for dioxin and furans and using methods different from those approved in the Endangerment Assessment. EPA then used this recalculated risk as support for its decision to recommend total on-site incineration of all affected materials.

MMI cannot understand the Agency's unilateral decision to recalculate the risk assessment figures based upon the new TEF values and changed methodology without notice or meaningful opportunity for comment by MMI. If MMI had received notice and opportunity to comment, the company could have explored the factual basis and scientific reasoning relied upon by the Agency to develop the new risk values. As matters now stand, however, there is no factual basis in the Administrative Record to support the newly calculated risks. Nor is there any explanation of the scientific reasoning upon which the revised TEF values are based. Moreover, if MMI had received notice and an opportunity to comment on the Agency's decision to use the new TEF values and changed methodologies, the company could have addressed other newly emerging scientific and regulatory information related to the risk assessment calculations. For example, newly emerging scientific information indicates that, even if the new TEF values are accepted, counterbalancing adjustments should be made to reflect more current understanding of the limited uptake and bioavailability of dioxins. EPA's unilateral decision to recalculate the risk estimate using new TEF values and changed methodology, but without using the comparable new information regarding uptake and bioavailability, is critical because the resulting recalculations erroneously overestimate risk factors for the Arkwood site.

MMI retained an independent consultant, McLaren/Hart, to analyze the quantitative impact of the more current scientific information regarding uptake and bioavailability of dioxin, and to review both the Endangerment Assessment and the EPA recalculation of risk. McLaren/Hart's analysis assumed, without change,

the Agency's new TEF values for the octachlorodibenzo-p-dioxin, but the analysis also incorporated reasonable adjustments to reflect the more current understanding of the limited uptake and bioavailability of dioxins. This analysis, which is included in the attached discussion, arrives at new risk estimates that are two orders of magnitude lower than the estimates reached in the original Endangerment Assessment. Finally, EPA disregarded site specific conditions when proposing the dioxin cleanup levels of 20 ppb (as 2,3,7,8 equivalents).

EPA's failure to give notice and an opportunity to comment on its decision to recalculate the Arkwood risk estimates based upon the new TEF values is compounded by the new TEF's not having undergone formal adoption through Agency rulemaking or any comparable legal process. In the absence of any such process, the new TEF values are not entitled to any greater weight or deference than any other scientific theory or estimate. It is MMI's understanding, however, that EPA has adopted a policy of applying the new TEF values without exception in order to promote the goal of interagency and international uniformity. While the goal of uniformity may have general merit, it should not be applied in a manner that deprives affected parties of any opportunity to comment upon the specific application in question.

2. <u>The Water Line That Is Being Installed Eliminates Any Risk Due to Possible Future Contamination of Nearby Domestic Wells</u>.

The principal potential and perceived risk from the Arkwood site--to the degree there is any risk at all--arises from the potential for future contamination of domestic wells immediately downgradient from the site. Although none of the wells sampled during the Remedial Investigation showed contamination related to the Arkwood site, the potential and perceived risk for exposure through future use of water from domestic wells will be eliminated by the extension of the Omaha public water system to all affected residences. As reflected in the attached materials, extension of the municipal water lines is already underway. Thus, none of the remedial alternatives can be justified by risks related to the potential for future contamination of domestic wells.

3. <u>Alternative D - Incinerate Sludges/Consolidate and Cap Affected Soils - Fully Satisfies All Significant Remedial Concerns</u>.

Remedial Alternative D in the Feasibility Study proposed off-site incineration of the more heavily affected materials at the site and consolidation and capping of the lesser affected

soils. This alternative would achieve the permanent destruction, through off-site incineration, of approximately 40% of the contaminants at the site. The balance of the contaminants strongly adhere to the soils. Consolidating and capping these materials would effectively eliminate the potential for further exposure by precluding direct physical contact and reducing infiltration to negligible levels. EPA rejected this alternative on the theory that the capped material could be subject to catastrophic failure in the event a sinkhole formed in the area. The only support for this notion of catastrophic sinkhole failure is an internal Agency memorandum prepared well after the Remedial Investigation and Feasibility Study reports were completed and approved by EPA. MMI asked the independent consultant that had prepared the RI/FS reports, ERM-Southwest, to review the memorandum and comment on the Agency concerns. ERM's comments are set forth in the attached materials. ERM concludes, in essence, that EPA's concerns are not consistent with the accumulated knowledge we have regarding the geology of the Arkwood site and vicinity. Moreover, ERM concludes that the Agency concerns regarding catastrophic encapsulation remedy failure due to sinkhole development could be addressed by appropriate design and monitoring techniques.

4. <u>EPA Summarily Rejected A More Cost Effective Remedy Recently Proposed At A Wood Treatment Site in Region 6</u>.

The Arkwood Feasibility Study included a Treatability Study which established that most of the contaminants reside in the finer fraction of soil particles, and that dramatic reductions in volume of contaminated materials can be achieved by sieve and wash techniques which separate the contaminated fines from the essentially uncontaminated rock fragments in the native soil. The Treatability Study also established that the fines could be biologically treated to concentrations at or below the PCP action levels proposed for the Arkwood site. EPA's Proposed Plan, however, rejected sieve and wash plus biological treatment, apparently on the ground that biological treatment would not destroy the trace levels of dioxin found in some of the waste. MMI believes that the Agency's reasoning in this regard is incorrect. Even if one ignores the dioxins, EPA's Proposed Plan recognizes that bioremediation "destroys most of the contamination found onsite[.]" (Proposed Plan of Action, p. 6.) Effective treatment of the dioxin could also be achieved by including stabilization as an element of this remedy for the fine-grained material after the biological treatment step. It is well established that stabilization is an effective treatment technology for dioxin. Indeed, the combination of bioremediation followed by stabilization was recently selected by EPA as the

preferred remedy in the Texarkana Wood Preserving Company Proposed Plan of Action. MMI respectfully submits that there is no basis in the record for rejecting at the Arkwood site the same remedial techniques--proven to be effective in a site-specific treatability study--which were approved by the Agency for comparable contaminants at the Texarkana Wood Preserving Company site.

5. Sieve and Wash Should Precede Any Treatment Technology Selected for the Arkwood Site.

The surficial residuum at the Arkwood site is primarily a red cherty clay. Visual observation of this site indicates that a majority of this material, by volume, consists of chert rocks. Composite sampling during the Treatability Study confirmed that approximately 70% of this material is greater than 12 mesh (0.055 inches) in size. This coarse material was almost entirely chert rock.

As noted above, the Treatability Study also established that a majority of the contaminants at the Arkwood site reside in the fine-grained material. Application of sieve and wash techniques separated these contaminated fines from the chert rocks. Samples analyzed during the Treatability Study indicated that use of sieve and wash techniques reduced the concentrations of PCP in the coarse materials below the proposed action levels, and reduced the concentration of carcinogenic PNA's below any reasonable cleanup level.

The results of the Treatability Study clearly indicate that sieve and wash is a cost-effective means of reducing the volume of contaminants to be dealt with. The sieve and wash process produces a relatively homogenous fine material, which is much more susceptible to effective treatment at reduced cost and reduced time for completion.

Based upon the results of the Treatability Study, MMI submits that sieve and wash must be included as a pre-treatment step before any treatment remedy that might be selected at the Arkwood site.

6. Another Remedial Alternative Exists Which Would Be Preferable to the Proposed Remedy.

EPA's preference for on-site incineration represents a dramatic departure from the position expressed by the Agency at a public hearing in Omaha on February 12, 1990. The Regional Project Manager for the site stated that the possibility of on-

site incineration of any material was "very small, very small." (Transcript of February 12, 1990 Public Hearing, p. 71.)

EPA's change of position apparently arises out of two new considerations: the recent revision in the Toxicity Equivalency Factors for dioxins and the recent concern about catastrophic encapsulation remedy failure due to sinkhole development. For the reasons explained elsewhere in these comments, MMI believes that neither of these new concerns has technical merit or factual basis as related to the Arkwood site. Assuming, however, that the Agency persists in its recently developed concerns, MMI submits that sieve and wash followed by in situ vitrification is a remedial alternative which would fully satisfy the Agency's concerns. This alternative would also equal or exceed the technical objectives of total on-site incineration at far less cost and with far greater likelihood of community acceptance.

MMI recognizes that in situ vitrification was dismissed from further consideration during the early stages of the Feasibility Study due to concerns that the technology was not then commercially practicable. Since that time, however, significant advances have been made in practical applications of the technology, and in commercial availability of a vendor capable of actual remedy implementation. Based on current knowledge, in situ vitrification of the fine-grained materials coupled with the sieve and wash techniques described in the Feasibility Study, represents a viable remedial option for the Arkwood site. For precedent, we refer you to the recent Record of Decision at the Crab Orchard National Wildlife Site.

To the extent that EPA feels it may lack information sufficient to select soil washing/in situ vitrification as a remedy alternative, MMI requests that the Agency defer final remedy selection and allow MMI to conduct a Focused Feasibility Study of a remedy based upon sieve and wash technologies plus in situ vitrification. Such a Focused Feasibility Study could include treatability studies with bench scale demonstration of the feasibility of treating materials from the Arkwood site by in situ vitrification technology.

MMI recognizes that EPA does not ordinarily revisit completed feasibility studies on the basis of subsequent developments in treatment technologies. MMI submits that further study is warranted in this case due to the unexpected concern about dioxin and catastropic sinkhole development expressed after completion and approval of the Remedial Investigation and Feasibility Study reports. There are no imminent hazards or other site conditions that would warrant or require immediate action. Moreover, MMI has cooperated fully with EPA throughout

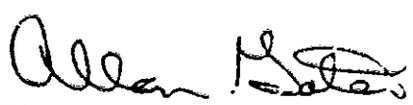
the process of site investigation and remedy selection. This long pattern of cooperation by MMI deserves reciprocation by the Agency, and it should mitigate any Agency concern that granting MMI's request for further study might be cited by PRPs in other sites as a precedent for requests that might frustrate the orderly CERCLA process.

CONCLUSION

As set forth in the more detailed supporting documents which follow, MMI opposes EPA's proposal for total on-site incineration of all affected materials at the Arkwood site. MMI believes that Alternative D is the most appropriate remedy for the site. The objections that EPA has raised to Alternative D can be resolved by the addition of stabilization and stormwater controls to the remedy. MMI disagrees with the newly-developed concerns which have prompted the Agency to select total on-site incineration. If, however, the Agency remains convinced on these points, MMI requests the opportunity to conduct a Focused Feasibility Study on a remedy option based upon sieve and wash plus in situ vitrification. MMI believes that such a remedy would be far superior to total on-site incineration. If the Agency denies MMI's request to conduct a Focused Feasibility Study, the company believes that any treatment alternative--including even on-site incineration--should be preceded by application of the demonstrated sieve and wash technology.

Respectfully submitted,

MITCHELL, WILLIAMS, SELIG & TUCKER

By 
Allan Gates

Counsel for Mass Merchandisers, Inc.

AG:gs

cc: Arkansas Department of Pollution Control and Ecology

TECHNICAL SUPPORT FOR PUBLIC COMMENTS OF MASS MERCHANDISERS, INC.

Arkwood, Inc. Site
Omaha, Arkansas

Prepared For:

Mass Merchandisers, Inc.

September 14, 1990

Prepared By:

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TABLE OF CONTENTS

Section		Page
I	INTRODUCTION	1
II	ALTERNATIVE D - INCINERATE SLUDGES/CONSOLIDATE AND CAP-IN-PLACE AFFECTED SOILS	2
III	ALTERNATIVE D+2	10
IV	GENERAL ASSESSMENT OF EPA'S PREFERRED SOILS REMEDY	15
	A. General Considerations	15
	B. Implementability	15
	C. Cost-Effectiveness	16
	D. Community Acceptance	16
	E. Summary	17
V	SIEVE AND WASH	18
	A. Background	18
	B. Particle Size Analysis of Residuum	18
	C. A Well Established System	17
	D. Applicability of Soil Washing to the Arkwood, Inc. Site Residuum	23
VI	ALTERNATE TREATMENT OPTIONS	24
	A. Soil Washing/On-Site Incineration	24
	B. Soil Washing/Biological Treatment/Stabilization	24
	1. Introduction	24
	2. Treatability Study	26
	a. Soil Washing	26
	b. Biological Treatment	26
	c. Conclusions from the Treatability Study	27
	3. Developing an Innovative Technology Design for Arkwood, Inc. Site	28
	a. Soil Washing	29
	b. Bioremediation	29
	c. Stabilization	30
	d. Conceptual Treatment Process	30
	4. Conclusions	33
	C. Feasibility Study of Soil Washing/In Situ Vitrification	35
	1. Introduction	35
	2. Applicability of ISV to the Arkwood, Inc. Site	39
	3. Advantages of ISV	42
	4. ISV Precedents	42
	5. Implementability of ISV	45
	6. Comparison of Soil Washing/ISV to Alternative H	45
	7. Recent Improvements to ISV	46

008661

TABLE OF CONTENTS (Cont'd)

Section		Page
VII	EVALUATION OF EPA'S CANCER RISK ASSESSMENT FOR THE ARKWOOD, INC. SITE	49
	A. Derivation of Dioxin Concentrations	49
	B. Procedure for Assessing Total Risk	50
	C. Target Carcinogenic Risk Level	50
	D. Conclusions	51
	E. Supplemental Materials	51
VIII	ADMINISTRATIVE RECORD	53
	A. Incomplete Record	53
	B. EPA's Proposed Plan of Action	58
	C. Public Meetings	59

APPENDICES

APPENDIX A - Evaluation of the 1989 Endangerment Assessment for Arkwood, Inc. Site

APPENDIX B - Review and Critique of the Scientific Basis of the TEF for OCDD

APPENDIX C - Dioxin Clean-up Goals for Arkwood, Inc. Site

APPENDIX D - Alternatives to Incineration for Soils Containing Greater than 20 ppb Dioxin

APPENDIX E - Calculation of Preliminary Health-Based Soil Cleanup Levels for Polynuclear Aromatic Hydrocarbons (PAHs) at the Arkwood Site in Omaha, Arkansas

APPENDIX F - Alternative I - Sieve and Wash/In Situ Vitrification

APPENDIX G - Transcript of February 12, 1990 Public Meeting in Omaha, Arkansas

List of Figures

		Page
Figure 1 -	Generic Cap Cross-Section	3
Figure 2 -	Alt. D+2 - Consolidation, Stabilization and Cap-in-Place	11
Figure 3 -	Conceptual Process to Treat Arkwood Soil	31
Figure 4 -	Flowsheet: Proposed Treatment Process for the Arkwood, Inc. Site	32
Figure 5 -	Proposed Treatment Process Flowsheet Arkwood, Inc. Site	34
Figure 6 -	ISV Staged In Situ Application Concept for the Arkwood Inc. Site	36

008663

TABLE OF CONTENTS (Cont'd)

List of Tables

	Page
Table 1 - Alternative D+2	13
Table 2 - Soil Sieving	19
Table 3 - Sieve and Wash/Incinerate Sludges and Affected Soil Fines On Site	25
Table 4 - Waste Materials that have been Processed in ISV Tests	37
Table 5 - Typical Organic Destruction/Removal Efficiencies	38
Table 6 - Benefits of >12 Mesh Fraction Removal	40
Table 7 - Comparison of ISV to Incineration	47

008664

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HAND DELIVERY

Re: Comments of Mass Merchandisers, Inc. on EPA's Proposed Plan of Action for the Arkwood, Inc. Site, Omaha, Arkansas

Dear Ms. Greeney:

MMI's comments on the EPA Proposed Plan of Action for the Arkwood site notes that there have been certain omissions from the the Administrative Record. MMI has asked that these omissions should be corrected by addition of the materials to the final Administrative Record. Also, attached you will find items which should be included in the Administrative Record as supplements to MMI's comments:

- A videotape of the February 12, 1990 EPA Public Meeting in Omaha, Arkansas. A transcript of this meeting is included as an appendix to the main comment submitted by MMI.

- A November 15, 1989 EPA memorandum from Kendall Brown to Larry Wright regarding the disposal of wood treating wastes from the Arkwood site.

- A letter dated July 24, 1990, from Raymond B. Harbison to Frank Conner, and a copy of Dr. Harbison's curriculum vitae. Dr. Harbison's letter was submitted as a portion of Frank Conner's statement at the July 26, 1990, Public Meeting and should have been included in the record of that meeting.

If you have any questions regarding these supplements to MMI's comments, please do not hesitate to call me.

008665

Very truly yours,

MITCHELL, WILLIAMS, SELIG & TUCKER

By Allan Gates
Allan Gates

AG:gs
Enclosures

cc: Arkansas Department of
Pollution Control & Ecology

008666

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION VI
1445 ROSS AVENUE, SUITE 1200
DALLAS, TEXAS 75202

NOV 15 1989

MEMORANDUM

SUBJECT: Disposal of Wood Treating Wastes from the Arkwood Inc., Superfund Site

FROM: Randall E. Brown, Chief
RCRA Enforcement Branch (6H-C)

TO: Larry Wright, Acting Chief
Superfund Enforcement Branch (6H-H)

This memorandum is in response to your request for assistance in determining whether the wastes at the Arkwood site are "dioxin" wastes which would require six nines destruction if incinerated.

In a letter dated March 4, 1986, from John Skinner (Office of Solid Waste) to Walter Talarek (American Wood Preserves Institute), an interpretation of the applicability of the dioxin rule to wastes generated at wood-treating facilities stated that most wastes from these operations would not be considered to be "dioxin" wastes unless such wastes met the specific listings of F021, F027, or F028 (see attachment).

In conversations between Ruth Izraeli and Jerry Truitt of my staff it appears that the waste materials at the Arkwood site would not be considered to be "dioxin" wastes. If you have any questions, please contact Jerry Truitt of my staff.

Attachment

008667

CURRICULUM VITAE

Raymond D. Harbison, M.S., Ph.D.
Professor and Director
Center for Environmental Toxicology
University of Florida

008668

CURRICULUM VITAE

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University of Iowa, Iowa City, Iowa - 1965 to 1967, M.S. (Pharmacology) -
Thesis: Induced hyperbilirubinemia and a quantitative method of analysis of diazotized bilirubin.

Ph.D. (Pharmacology/Toxicology) - Dissertation: Studies on the mechanism of teratogenic action and neonatal pharmacology of diphenylhydantoin.

SUMMARY OF EXPERIENCE

1989-Present
Director, Center for Environmental Toxicology, University of Florida.

1989-Present
Staff, St. Vincent Infirmary Medical Center, Little Rock, Arkansas.

1989 - Present
National Institute of Drug Abuse, Pharmacology II Study Review Group

1988-Present
Professor, Department of Pharmacology and Therapeutics, School of Medicine, University of Florida.

1988-Present
Professor, Department of Physiological Sciences, Health Science Center, University of Florida.

1987-Present
Clinical Professor, Department of Preventive Medicine, Medical College of Wisconsin, Milwaukee, Wisconsin.

1986-Present
National Institute of Environmental Health Sciences Study Review Group

1984-1985
Chairman, National Institute of Occupational Safety and Health Study Review Group

008669

1982-1986
Editonal Board, Fundamental and Applied Toxicology

1980-Present
Society of Toxicology Liaison with Teratology Society

1980-1988
Professor, Department of Pharmacology and Director, Interdisciplinary Toxicology Graduate Program, University of Arkansas for Medical Sciences.

1980-1984
National Institute of Occupational Safety and Health Study Review Committee

1979-Present
Editorial Board, Teratogenicity, Mutagenicity, Carcinogenicity

1977-1980
Director, Toxic Substance Control Laboratory and Associate Professor, Department of Pharmacology and Biochemistry, School of Medicine, Vanderbilt University, Nashville, Tennessee.

1977-1980
Professional Affairs Committee - American Society for Pharmacology and Experimental Therapeutics

1977-1980
National Institute on Drug Abuse - Review of DAWN (Drug Abuse Warning Network)

1977-1979
Graduate Education Committee, Vanderbilt University

1977-1978
National Research Council Committee to Review Scientific Program of National Center for Toxicological Research

1976-1980
Editonal Board, Environmental Health Sciences

1976-1977
National Academy of Science, Advisory Center for Toxicology - Revision of Toxicity Testing Procedures for Consumer Protection Agency

1975-1976
Technical Committee of the Society of Toxicology, Chairman

1975-1976
National Institute on Drug Abuse Center Review Committee

1974-Present
Editonal Board, International Journal of Addictive Diseases

1974-1979
Vanderbilt Medical Center, Animal Care Committee

008670

1974-1978
Consultant, U.S. Congressional Committee on Safety Assessment of Chemical Additives and Drugs

1974-1975
Standing Policy Committee on Biomedical Sciences, Vanderbilt School of Medicine; National Institute on Drug Abuse, Clinical Behavioral Review Committee

1974-1975
Co-Chairman, Technical Committee of the Society of Toxicology

1972-1976
Assistant Professor of Pharmacology and Biochemistry, Vanderbilt University School of Medicine

1971-1975
National Institutes of Mental Health-Narcotic Addiction and Drug Abuse Review Committee, Biomedical-Pharmacology-Toxicology

1971-1972
Assistant Professor, Department of Pharmacology, Tulane Medical School

1971-1972
Director of Teratology Section, Laboratory of Environmental Health, Department of Medicine, School of Medicine, Tulane University

1969-1970
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1965-1969
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Sigma Xi (Promotion of Research in Science)
American Association for the Advancement of Science
Teratology Society
Society of Toxicology
American Society for Pharmacology and Experimental Therapeutics
New York Academy of Science
Society for Risk Analysis

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Society of Toxicology Achievement Award

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Certified in General Toxicology

008671

INDUSTRIAL EXPERIENCE

- Occidental Oil - Worker safety in oil shale production
- Shell Development Corporation - Pesticide use and safety
- Petrolite Corporation - Health assessment of waste incineration methods
- Monsanto Corporation - Chemical mutagenesis and the workplace, environmental assessment of PCB pollution
- American Academy of Industrial Medicine - Women in the workplace
- Tennessee Occupational Safety Health Administration - Industrial toxicology training course
- Society Organic Chemical Manufacturing Association - Chemical carcinogenesis
- Sanitary Corporation of America - Worker safety at industrial residue landfill sites
- Ethyl Corporation - Chemical-induced mutagenesis and teratogenesis in the workplace
- State of Kentucky Bureau of Natural Resources - Toxicology training course for solid waste management personnel
- U.S. Environmental Protection Agency - Chemistry and toxicology of hazardous materials training course for spill management personnel
- Ecology and Environment, Inc. - Health and safety program advisor
- Texaco - Toxicology consultant
- Hooker Chemical Company - Evaluation of health effects at Love Canal
- Chemical Manufacturers Association - Technical review of health effects of PCBs
- American Petroleum Institute - Comments to EPA concerning Resource Conservation Recovery Act
- IBM Corporation - Reproductive hazard assessment
- U.S. Environmental Protection Agency - Rebuttable presumption review of pesticides (FIFRA)
- United States Department of Agriculture - Review of aerial pesticide applications
- State of Georgia Department of Environmental Protection - Toxicology training course for emergency environmental incident management
- State of North Carolina Environmental Resource Management Division - Toxicology training course for environmental management and public health personnel

008672

- Exxon - Review of teratogenic hazard of benzene exposure
- U.S. Department of Justice - Evaluation of environmental and public health problems associated with Price Landfill, Atlantic City, New Jersey
- U.S. Department of Justice - Evaluation of environmental and public health problems associated with Bridgeport Oil and Rental contamination of the Delaware River, Bridgeport, New Jersey
- U.S. Environmental Protection Agency - Evaluation of the environmental impact of dredging of the Hudson River for PCBs, New York
- U.S. Environmental Protection Agency - Evaluation of environmental and public health problems associated with LiPari Landfill in New Jersey
- Velsicol Chemical Corporation - Evaluation of health problems associated with Hardeman County Landfill
- U.S. Environmental Protection Agency - Review of environmental and public health information associated with Denny Farm Site, Verona, Missouri
- U.S. Environmental Protection Agency - Review of environmental and public health information associated with Rose Park Landfill, Salt Lake City, Utah
- U.S. Environmental Protection Agency - Evaluation of environmental and public health problems associated with Taylor Road Landfill, Tampa, Florida
- Dow Chemical Company - Evaluation of health problems associated with a degreasing operation in Tyler, Texas
- United States Environmental Protection Agency - Training course director for Toxicology and Risk Assessment for eight southeastern states. (Alabama, Florida, Georgia, Kentucky, Mississippi, North Carolina, South Carolina, Tennessee
- Monsanto Chemical Company - Evaluation of adverse health effects associated with PCB contamination of feed grain in Michigan
- American Bar Association - Short course concerning the role of expert testimony in environmental litigations.

008673

GRANT SUPPORT

Effect of Environmental Toxicants on Perinatal Development	ES00782	1970-1982
Effect of Marijuana on Perinatal Development	DA00141	1971-1974
Schleider Foundation Developmental Toxicology		1970-1972
Clinical Pharmacology - Toxicology Center	GM15431	1975-1981
Environmental Toxicology Center	ES00267	1972-1981
Synthesis and Study of New Chelating Agents	ES01018	1975-1981
Life Insurance Research Fund		1970-1981
National Conference on Control of Hazardous Materials Spills	EPA	1977-1978
Wampole Laboratories - Development of a Prenatal Diagnostic Aid for Neurotube Defects		1979-1981
Introduction to Hazardous Materials Incidence Responses and Environmental Hazards Evaluation	EPA	1980-1981
Environmental Toxicant Effects on Perinatal Development	ES02824	1981 1986
Study of the Reproductive Toxicity of Selected Chemicals	NF	1985-1987
Studies of Chemical-Induced Toxicity and Stress	ES05216	1988-1993
Studies of Isomer Specific PCB-Induced Toxicity	*Cooperative Agreement	1989-1991

* Department of Environmental and Community Medicine UMDNJ - Robert Wood Johnson Medical School Piscataway, New Jersey

ES - National Institute of Environmental Health Sciences
DA - National Institute on Drug Abuse
GM - National Institute of General Medical Sciences
EPA - United States Environmental Protection Agency
NF - National Foundation March of Dimes

008674

TEACHING EXPERIENCE

Medical Toxicology	Vanderbilt Medical Center Second Year Medical Pharmacology
Developmental Pharmacology	Vanderbilt Medical Center Second Year Medical Pharmacology
Drug Metabolism	Two Hour Graduate Level Course Vanderbilt Medical Center
Toxicology	Two Hour Graduate Level Course Vanderbilt Medical Center
Medical Toxicology	Second Year Medical Pharmacology University of Arkansas for Medical Sciences
Advanced Toxicology	Two Hour Graduate Level Course University of Arkansas for Medical Sciences
Oncology	Two Hour Graduate Level Course University of Arkansas for Medical Sciences
Medical Toxicology	Second Year Medical Pharmacology University of Florida, School of Medicine
Mechanism of Chemical-Induced Toxicity	Two Hour Graduate Level Course University of Florida, School of Medicine

Continuing Education:

National Hazardous Materials Training Course	Eight Hours (Toxicology)
Hazardous Waste Management	Four Hours (Toxicology)
Toxic Substance Control	Eight Hours (Toxicology)
Environmental Protection Agency, Region IV, Health and Safety Training School	Six Hours (Toxicology)
Industrial Toxicology	Five Hours (Carcinogenesis, Mutagenesis, Teratogenesis)
Forensic Medicine	Three Hour (Toxicology)

008675

GRADUATE TRAINING - PREDOCTORAL

	Year Degree Conferred	Present Address
Bernardo Mantilla-Plata, Ph.D.	1972	University of Antioqua Department of Toxicology Medellin, Columbia
Michael Stevens, Ph.D.	1973	Monsanto Chemical Company Toxicology Department St. Louis, Missouri
Richard W. Freeman, Ph.D.	1980	TERRA, Inc. Tallahassee, Florida
Michael E. Fant, M.D., Ph.D	1980	Department of Pediatrics Southwest Medical School Dallas, Texas
Adeline Smith, Ph.D.	1982	National Institute of Health Div. of Molecular Toxicology Bethesda, Maryland
James Jernigan, Ph.D.	1983	U.S. Army Biomedical Research Command Dugway, Utah
Christopher Teaf, Ph.D.	1985	Florida State University Institute of Science and Public Affairs Tallahassee, Florida
Felix Adatsi, Ph.D.	1986	Department of Natural Resources Surface Water Quality Div. State of Michigan Lansing, Michigan
M. Ann Clevenger, Ph.D.	1987	EPA Washington, D.C.
Glenn C. Millner, Ph.D.	1987	Terra, Inc. Little Rock, Arkansas
Henry F. Simmons, M.D., Ph.D.	1988	Division of Clinical Toxicology University of Arkansas for Medical Sci. Little Rock, Arkansas
Mary Alice Smith	1989	School of Medicine Emory University Atlanta, Georgia
Hudson K. Bates	1989	Research Triangle Institute Research Triangle Park, NC

008676

GRADUATE TRAINING - POSTDOCTORAL

	Years of Study	Present Address
Michael Evans, Ph.D.	1973-1976	University of Illinois Dept. of Pharmacology Chicago, Illinois
Chandrahar Dwivedi, Ph.D.	1973-1976	Meharry Medical College Dept. of Pediatrics Nashville, Tennessee
Richard P. Koshakji, Ph.D.	1973-1976	Tennessee Neurophysiciatric Institute Nashville, Tennessee
James S. MacDonald	1975-1977	Merck Institute for Therapeutic Research West Point, Pennsylvania
Daniel Goodman, Ph.D.	1978-1980	Univ. of Arkansas Med. Sci. Div. of Interdis. Toxicology Little Rock, Arkansas
Robert C. James, Ph.D.	1979-1981	Univ. of Florida Ctr. for Environ. Toxicology Gainesville, Florida
Peter Wells, Pharm.D.	1979-1981	University of Toronto College of Pharmacy Toronto, Ontario, Canada
Shahata El-Sewedy, Ph.D.	1982-1983	University of Alexandria Medical Research Institute Alexandria, Egypt
Syed F. Ali, Ph.D.	1982-1983	Univ. of Arkansas Med. Sci. Dept. of Biochemistry Little Rock, Arkansas

008677

PUBLICATIONS

1. Harbison, R.D., R.C. Boerth and J.L. Spratt. Quantitative determination of free and conjugated bilirubin by diazo coupling and a liquid-extraction and column-chromatographic technique. Biochem. J. 104:46c-47c, 1967.

2. Harbison, R.D. and J.L. Spratt. Novobiocin-induced hyperbilirubinemia and its reduction by phenobarbital pretreatment. Toxicol. Appl. Pharmacol. 11:257-263, 1967.

3. Harbison, R.D. and J.L. Spratt. Disappearance of plasma bilirubin fractions in the rat after phenobarbital. Arch. Int. Pharmacodyn. 172(1):32-36, 1968.

4. Harbison, R.D. and B.A. Becker. Barbiturate mortality in hypothyroid and hyperthyroid rats. J. Pharm. Sci. 58(2):183-185, 1969.

5. Harbison, R.D., C.D. Klassen and B.A. Becker. Hemodynamics of the isolated perfused liver of hypothyroid and hyperthyroid rats. Proc. Soc. Exper. Biol. Med. 132:96-99, 1969.

6. Harbison, R.D. and B.A. Becker. Relation of dosage and time of administration of diphenylhydantoin and its teratogenic effect in mice. Teratology 2:305-311, 1969.

7. Eling, T.E., R.D. Harbison, B.A. Becker and J.R. Fouts. Diphenylhydantoin effect on neonatal and adult rat hepatic drug metabolism. J. Pharmacol. Exptl. Therap. 171:127-137, 1970.

8. Eling, T.E., R.D. Harbison, B.A. Becker and J.R. Fouts. Kinetic changes in microsomal drug metabolism with age and diphenylhydantoin treatment. Europ. J. Pharmacol. 11:101-108, 1970.

9. Harbison, R.D. and B.A. Becker. Effect of phenobarbital and SKF 525-A pretreatment on diphenylhydantoin teratogenicity in mice. J. Pharmacol. Exptl. Therap. 171:283-288, 1970.

10. Harbison, R.D. and B.A. Becker. Effect of phenobarbital or SKF 525-A pretreatment on diphenylhydantoin deposition in pregnant mice. Toxicol. Appl. Pharmcol. 20:573-581, 1971.

11. Harbison, R.D. and B. Mantilla-Plata. Prenatal toxicity, maternal distribution and placental transfer of tetrahydrocannabinol. J. Pharmacol. Exptl. Therap. 180(2):446-453, 1972.

12. Harbison, R.D. and B.A. Becker. Diphenylhydantoin teratogenicity in rats. Toxicol. Appl. Pharmacol. 22:193-200, 1972.

13. Wilson, B.J. and R.D. Harbison. Rubratoxins. J. Am. Veterinary Med. Assoc. 163(11):1274-1276, 1973.

14. Koshakji, R.P., B.J. Wilson and R.D. Harbison. Effect of rubratoxin B on prenatal growth and development in mice. Res. Comm. Chem. Pathol. Pharmacol. 5:584-593, 1973.

008678

15. Koshakji, R.P., J. Cole and R.D. Harbison. Influence of injection volume on parathion toxicity and plasma and brain cholinesterase inhibition. Res. Comm. Chem. Pathol. Pharmacol. 6(2):677-687, 1973.

16. Mantilla-Plata, B. and R.D. Harbison. Effect of phenobarbital and SKF 525-A pretreatment, sex, liver injury and vehicle on Δ^9 tetrahydrocannabinol toxicity. Toxicol. Appl. Pharmacol. 27:123-130, 1974.

17. Jones, M.M. and R.D. Harbison. Phthalyltetrathioacetic acid (PTTA). A new chelating agent. Res. Comm. Chem. Pathol. Pharmacol. 7:390-398, 1974.

18. Stevens, M.W. and R.D. Harbison. Placental transfer of diphenylhydantoin: Effects of species, gestation age, and route of administration. Teratology 9:317-326, 1974.

19. Harbison, R.D. and B.A. Becker. Comparative teratogenicity of diphenylhydantoin and metabolites in mice. Teratology 10(3):237-242, 1974.

20. Dwivedi, C., R.D. Harbison, B. Ali and S. Parmar. Synthesis of substituted anilino-[3-methoxy-4-(4-arylthiosemicarbazidecarbonylmethyleneoxyl)] benzylidenes: Correlation between anticonvulsant activity and monoamine oxidase inhibitory and antihemolytic properties. J. Pharmaceut. Sci. 63(7):1124-1128, 1974.

21. Parmar, S.S., B.R. Pandey, C. Dwivedi and R.D. Harbison. Anticonvulsant activity and monoamine oxidase inhibitory properties of 1,3,5-trisubstituted pyrazolines. J. Pharmaceut. Sci. 63(7):1152-1155, 1974.

22. Koshakji, R.P., B.V. Rama Sastry and R.D. Harbison. Studies on the levels and nature of cholinesterase in human and mouse placenta. Res. Comm. Chem. Path. Pharmacol. 9(2):687-692, 1974.

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33. Harbison, R.D. Comparative toxicity of some selected pesticides in neonatal and adult rats. Toxicol. Appl. Pharmacol. 32(2):443-455, 1975.

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008680

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44. MacDonald, J.S. and R.D. Harbison. Methyl mercury-induced encephalopathy in mice. Toxicol. Appl. Pharmacol. 39:195-205, 1977.

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49. Harbison, R.D., M.M. Jones, J.S. MacDonald, T.H. Pratt and R.L. Coates. Synthesis and pharmacological study of a polymer which selectively binds mercury. Toxicol. Appl. Pharmacol. 42:445-454, 1977.

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56. Evans, M.A. and R.D. Harbison. Cocaine-induced hepatotoxicity in mice. Toxicol. Appl. Pharmacol. 45:739-754, 1978.

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008686

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008694

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July 24, 1990

Mr. Frank Conner
President and Chief Operating Officer
Mass Merchandisers, Inc.
Post Office Box 790
Harrison, Arkansas 72601

Dear Mr. Conner:

As you requested, I have begun an analysis of EPA's assessment of public health risks associated with the Arkwood site. I have also reviewed the dioxin sample results relied upon by EPA in support of its recent announcement that it proposes on-site incineration as a part of the remedial action for the site. It is my opinion that there is no significant risk to human health from the levels of dioxin reported in the sample results that I have reviewed. This is true for several reasons.

First, the most toxic form of dioxin -- the 2,3,7,8 isomer of tetrachlorodibenzo-para-dioxin or TCDD--was not detected in any of the sample results that I have seen. This is not particularly surprising since TCDD is rarely found in wood-treating chemicals.

Second, the type of dioxin detected in the samples relied upon by EPA — particularly the hepta-, hexa-, and octa- dioxins — are hundreds to thousands of times less toxic than the tetrachloro dioxin or TCDD. We are now finding that these less toxic forms of dioxin are widely distributed throughout our everyday environment, with no observed ill effects on humans.

Third, despite the enormous public attention that has been focused on dioxin in the past decade, there is a clear trend of scholarly opinion in the scientific community to the effect that the human health risks from exposure to dioxin have been greatly exaggerated in the past.

As you requested, I have begun a detailed examination of EPA's endangerment assessment calculations. I will submit a written analysis to you as soon as possible. Based on my initial review, I believe that EPA's Report overstates the risks to human health and that the current state of scientific knowledge does not support EPA's decision to propose on-site incineration at the Arkwood site.

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SECTION 1

TECHNICAL SUPPORT FOR PUBLIC COMMENTS
OF MASS MERCHANDISERS, INC.

Arkwood, Inc. Site
Omaha, Arkansas

I. INTRODUCTION

The following comments and discussions are presented in response to the EPA's July 1990 Proposed Plan of Action for the Arkwood, Inc. Superfund Site. This proposed plan identified the EPA's preferred option for addressing affected soils at the Arkwood, Inc. site in Omaha, Arkansas as on-site incineration of all sludges and affected soils above the action levels (approximately 20,960 cubic yards). In addition, the proposed plan included summaries of other alternatives for the site which were discussed in the Feasibility Study (FS) Report.

Three significant changes have occurred since the development of the FS Report. These are 1) the re-evaluation of dioxin concentration at the site using the most recent toxicity equivalency factors (TEFs); 2) concerns regarding catastrophic sinkhole development at the site; and 3) recent improvements to in situ vitrification (ISV) technology. These changes are addressed in the following comments as well as a re-evaluation of remedial alternatives for the site. ERM-Southwest appreciates this opportunity to submit new information for EPA's consideration before selecting the final soils remedy.

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SECTION 2

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II. ALTERNATIVE D - INCINERATE SLUDGE CONSOLIDATE AND CAP-IN-PLACE AFFECTED SOILS

As presented in the FS Report, Alternative D called for fencing of the site, existing structures to be removed, and railroad ditch and sinkhole sludges to be incinerated off site. The affected soils would then be excavated, consolidated and covered with a composite cap. This cap, as illustrated in Figure 1, is an engineered cap, at least five feet thick and containing a 30-mil high-density polyethylene liner and geofabric. The remainder of the site would be covered with a topsoil cap.

In the process of selecting its preferred remedial option, the EPA summarily dismissed Alternative D as unacceptable. The Agency's stated reason for rejecting this and any remedy involving on-site encapsulation (capping) of affected soils was the possibility of a catastrophic sinkhole collapse which would allow capped, affected soils to be flushed directly into the ground water system. A secondary reason appears to be based on the presence of certain isomers of dioxin. Dioxin issues are addressed in the Appendices.

The concern over the possibility of catastrophic sinkhole collapse seems to have been a consequence of a conversation between Mr. Brent Truskowski and Mr. Malcolm Field of EPA's Exposure Assessment Applications Branch in Washington, D.C. Documentation of this concern first appears in the Administrative Record as an internal memorandum by Mr. Field dated July 3, 1990. This concern was reiterated in the June 27, 1990 EPA memorandum from Mr. Allyn Davis to the Directors of the Office of Emergency and Remedial Response and Office of Waste Programs Enforcement stating that "the proposed incineration approach was considered appropriate because of the karst geology underlying the site". In the July 3, 1990 memorandum, Mr. Field stated that the possibility does exist that sinkhole development could cause contaminated site soils to be flushed directly into the aquifer system and that this possibility completely invalidates any remedial alternative which includes waste being retained in capped soils as a permanent solution. He adds that should a sinkhole form under the capped soils, the soils could be flushed into the uppermost aquifer at a rate of several feet per hour.

It should be noted that Mr. Field had never visited the site at the time he wrote the letter and was merely describing what he called probable effects based on his general knowledge of karst terrains. Without obtaining site-specific information, EPA concluded that all remedies involving on-site capping of affected soils were unacceptable. Furthermore, the Agency did so <u>after</u> the Remedial Investigation (RI) was concluded, thus foreclosing the collection of data to more precisely quantify the site-specific risk. However,

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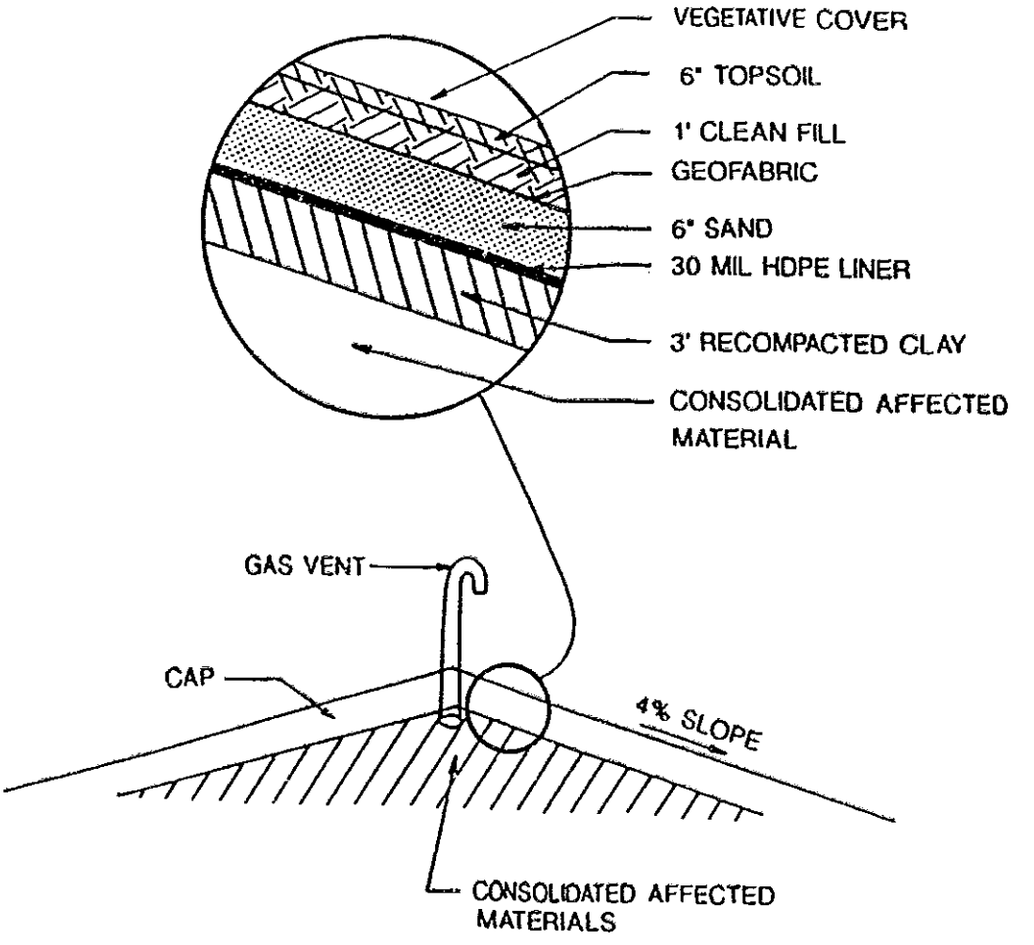

NOTE: SEE DESCRIPTIONS OF INDIVIDUAL ALTERNATIVES FOR SPECIFIC CAP LAYOUTS

ERM-Southwest, inc.
NEW ORLEANS, LOUISIANA HOUSTON, TEXAS

DATE 12/01/89 | W.O.NO. 9209A014X

FIGURE 1
GENERIC CAP CROSS-SECTION
Arkwood, Inc. Site
Omaha, Arkansas

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more than enough is known for ERM-Southwest to render the opinion that remedial options involving capping of affected soils should not be arbitrarily dismissed because of concern over a hypothetical sinkhole collapse beneath the cap. Mr. Field's use of the terminology "probable effects" is unfortunate since it denotes an effect that is likely to happen. In reality, Mr. Field has no basis for concluding that a sinkhole would "probably" develop beneath capped soils. We believe that the EPA's decision to dismiss all capping options because of the "probability" of catastrophic sinkhole collapse was based on an inadequate understanding of the site-specific geology and hydrogeology.

The lack of easily recognizable sinkhole features within the Arkwood, Inc. site study area was discussed in the RI Report. The scarcity of recognizable sinkholes is a result, in part, of the small size and subtle topographic expression that these features characteristically display in this particular karst terrain. Sinkhole is a general term that can apply to any location where water disappears or sinks below the ground surface. The term does not denote magnitude. The "sinkhole" referenced in the RI as existing near the wood treatment building has only a very small depression surrounding it and would more appropriately be described as an open, solutionally enlarged joint. This enlarged joint was small and stable enough that it was completely covered by a 10 x 10 foot concrete slab in 1982. To date, this slab is still providing an unbroken protective seal over the sinkhole, thus confirming that further development and subsidence of the opening has not occurred.

The intermittent losing stream segment being proposed as a potential input point for the first dye tracing study may be considered another "sinkhole" feature similar to the one described above. It seems to have a very small areal extent with little or no topographic expression. In the karst terrain surrounding the Arkwood, Inc. site, this type of sinkhole appears to be normal and is not indicative of a potential for a catastrophic sinkhole collapse. In fact, no example of a large, catastrophic sinkhole collapse is documented within this part of Arkansas including the Arkwood, Inc. site study area. This is further supported by recent interviews with a local bridge and highway engineer, local paving contractors, and with the line superintendent for the Union Pacific Railroad responsible for inspection and maintenance of the track passing the site. None of these individuals could recall any construction or maintenance problems created by sinkhole collapses. Based on observations made during the RI field work, the characteristic sinkhole in the study area would have a width of no more than a few feet and a shallow depth relative to the width. Unless induced by man-made features, not present at the Arkwood, Inc. site, development of these characteristic sinkholes would

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proceed significantly slower than the rapid catastrophic collapses which occasionally occur in metropolitan areas over karst regions in other parts of the United States. In terms of the scale and destructive potential, catastrophic sinkhole collapses in other karst terrains are not relevant to sinkhole development at the Arkwood, Inc. site.

As opposed to the geology of some karst terrains in other areas of the United States which are noted for catastrophic sinkhole development, with which Mr. Field may be familiar, there are certain characteristics of the Arkwood, Inc. site, site-specific geology which would present obstacles to catastrophic sinkhole collapse. The apparent lack of large solution cavities within the St. Joe Limestone and lack of large cavities within the overlying residuum virtually eliminates the possibility of catastrophic collapse occurrences. The fact that the limestone bedrock is close to the surface, covered by a relatively thin residuum layer, would also tend to limit the degree of slope retreat and decline should a small collapse feature develop. The primary mechanisms for sinkhole development at the Arkwood, Inc. site would seem to be the relatively slow piping of residuum sediments into the conduit system. Without a major conduit system, without a well-defined and integrated water table within the residuum and with the natural ground slope providing good runoff drainage, there is no mechanism to promote rapid movement of large volumes of soil. This accounts for the small size and subtle nature of the two known sinkholes on the site.

One of the primary prerequisites for sinkhole development is an adequate water supply. Typical causes of catastrophic sinkhole collapses are man-made; i.e., fluctuations of the water table due to pumping, broken water or sewer lines and concentration of runoff due to urbanization. This prerequisite is not present at the Arkwood, Inc. site. In addition, drainage controls would be instituted as part of any remedy that requires capping and long-term maintenance. Excessive surface runoff would be controlled so as to remove the prerequisite of an adequate water supply. Rainflow runoff would be routed around and not over the site. In the highly unlikely event that a sinkhole began developing under or close to capped soil, there would be no mechanism to promote rapid enlargement or to flush large quantities of soil into the ground water system. The small size of such a feature and the lack of a flushing mechanism would result in little, if any, release to the ground water system. It would definitely not represent a random, catastrophic event that would threaten public health or the environment and for which no solution existed. In the unlikely event that soil was released from beneath the capped area, the release would be very gradual and thus, it would represent a controllable event that can be planned and designed for, and for

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which corrective measures could be taken before public health or the environment were threatened.

Rejection of on-site capping of affected soils solely because of a concern over catastrophic sinkhole collapse presumes that no other options for control of sinkhole formation and collapse exist. However, a considerable amount of literature is available concerning the recognition, evaluation and subsequent reduction of risk due to sinkhole collapse and concerning the remediation of sinkholes should they occur. There is nothing in the Administrative Record for the Arkwood, Inc. site to indicate that the EPA consulted any of this literature before concluding that the possibility of catastrophic failure made Alternative D unacceptable. Our review of this literature assures us that utilization of applicable and appropriate geological and engineering controls can essentially negate any risk presented by sinkhole collapse.

With the RI closed, insufficient data are available with which to generate a precise quantification of the risks presented by sinkholes, but a sense for their small magnitude can be extrapolated by evaluating what <u>is known</u> about the site and the vicinity. The following facts and observations should be considered:

1. No catastrophic sinkhole collapses within this part of Arkansas, including the site study area, are documented.

2. Local highway, contractor and railroad representatives contacted have no record of adverse conditions resulting from sinkholes.

3. The immediate study area contains only two known "sinkholes", both of which are small, unobtrusive features and only one (the intermittent losing stream segment proposed as a potential dye input point) of which is actively connected with the ground water system.

4. Although all sinkholes have common elements, the details of their development can vary considerably between different karst terrains; i.e., the fact that large, catastrophic sinkhole collapses can rapidly develop in Florida does not mean that similar sinkholes will develop in north central Arkansas, in fact, site-specific data support the lack of potential for any catastrophic sinkhole collapse.

5. As part of any remedy involving capping, drainage controls can be instituted to remove the prerequisite for sinkhole development, namely, an adequate supply of water.

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6. All appropriate preconstruction and engineering controls would be utilized during any capping remedy to recognize, evaluate and reduce any risk of sinkhole collapse. Special engineering considerations can be instituted so that a cap would not fail if a sinkhole were to develop beneath it.

7. Assuming a sinkhole collapse did occur under capped soil, there would be no flushing mechanism by which large quantities of affected soil could be introduced into the ground water system.

8. In most catastrophic sinkhole collapses, deeper soils are first introduced into the ground water system. Surficial soils (in this case, capped soils) would, in all probability, be suspended above the ground water flow regime for a sufficient period to allow their removal and remediation of the collapse area.

The EPA had numerous opportunities to express concerns about sinkhole development in the area dating back to 1986 when the RI/FS Work Plan was first developed. In addition, during the RI/FS in which field activities and numerous drafts were reviewed by EPA and their consultants, no concern was ever expressed about sinkhole development as a problem relative to site remediation. Therefore, no site-specific information was provided to the Agency for decision-making purposes, other than the cursory discussion in the RI Report concerning the on-site sinkhole and the lack of other easily recognizable sinkholes in the area. This information was apparently not considered and no additional data sought as the Agency appeared to rely entirely upon Mr. Field's remarks to invalidate remedial alternatives deemed by ERM-Southwest to be adequately protective of human health and the environment. In our view, it would be arbitrary to select a soils remedy without careful consideration of the site-specific data refuting EPA's concerns, contained here. Obviously, this data would have been provided during the RI if EPA's concern had been expressed.

It is believed that sufficient information exists to establish that the potential for catastrophic sinkhole development at the Arkwood, Inc. site is extremely low. If a sinkhole of the type and size present in the area were to develop under capped soils, the small size of the feature and the lack of a flushing mechanism would make it easy to remediate the problem, thus preventing any unacceptable threat to human health and the environment.

If the EPA's recently stated objection to on-site consolidation and capping is disregarded due to lack of site-specific relevance, Alternative D represents the best option for protecting human health and the environment, both in terms of short- and long-term

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effectiveness. It continues to be ERM-Southwest's opinion (see earlier draft of the FS Report) that Alternative D is the best soils remedy for the Arkwood, Inc. site. It is easily implementable and is much more cost-effective than total incineration. In support of Alternative D, the following additional items should be considered:

1. A properly engineered and maintained cap would virtually eliminate the infiltration of precipitation, thus preventing the transport of constituents to the ground water. The potential for constituent migration would be further mitigated by the strong adherence of wood treatment compounds to soil particles. This relative immobility of constituents was demonstrated during the Treatability Study.

2. The greatest risk (see Endangerment Assessment) at the Arkwood, Inc. site is associated with direct soil contact. Direct contact would be eliminated by consolidation and capping, thus effectively eliminating risk at the site. This is an accepted and proven technology, the effectiveness of which would not be altered by sinkhole development.

3. To further alleviate future concerns regarding off-site migration of affected ground water, MMI voluntarily offered to pay for installation of a city water line running a mile and a half down the valley from the Arkwood, Inc. site. MMI will also conduct long-term monitoring of selected wells and springs. Therefore, the health of individuals living down the valley from the site will be protected while monitoring for the highly unlikely event of a release to the ground water of material stored beneath the cap. The extension of the Omaha city water line to residents immediately downgradient from the Arkwood, Inc. site is underway. The work will be funded by Mass Merchandisers, Inc., and the contracting will be undertaken by the City of Omaha. Formal approval for the extension was granted by a resolution adopted by the Omaha City Council on August 29, 1990. The resolution calls for a 4-inch water line along Cricket Road to end at the Willie Tate residence and a 1-inch line from the Omaha School to the Rayford Duggen residence. The extension of the Omaha city water line will provide city water to all those residents who have wells included on the testing schedule. Phase I of the City's contract will be an on-site review and preliminary cost estimate by the City of Omaha's consulting engineers. This phase is expected to be completed by September 27, 1990. As soon as cost estimates are completed, engineering drawings will be submitted to the State Health Department for approval and construction can begin.

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4. As part of Alternative D, highly affected soils and sludges would be incinerated off-site, thus destroying approximately 40% of the known wood treatment compounds at the site. The volume of material to be capped on-site would be reduced and the most highly affected and toxic materials would be completely removed. Therefore, SARA requirements are met in all material respects by destruction of 40% of the mass of contaminants and the decrease in mobility by capping.

When Alternative D was originally evaluated in the FS, it emerged as a workable and cost-effective option for remediating the Arkwood, Inc. site. It met or exceeded all of the evaluation criteria including protection of human health and the environment. The EPA, in its discussion of the alternatives reached the same basic conclusion with the exception that it considered long-term effectiveness and permanence to be poor because of the sinkhole issue. However, as discussed above, the stated reason for rejecting remedies utilizing consolidate and cap, and in particular Alternative D, has no site-specific validity.

Consolidate and cap-in-place has been used in the past at other Superfund sites. For example, within EPA Region 6, it has been approved for use at the Mid-South Wood site in Polk County, Arkansas.

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SECTION 3

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III. ALTERNATIVE D+2

Nevertheless, Alternative D+2 is herein provided as a means of enhancing Alternative D in order to remove any possible Agency concern about catastrophic sinkhole development. D+2 adds supplemental engineering controls and solidification of the affected soils. For ease of comparison with other alternatives, the format used to present this alternative is the same as that used in the FS Report.

Alternative D+2 - Incinerate Sludges/Consolidate and Stabilize Soils/Cap-in-Place Affected Soil and Provide Stormwater Controls

Description

The proposed remedial alternative chosen by the EPA, on-site incineration of sludges and soils, is apparently based on concerns about the karst geology and the presence of certain isomers of dioxin. The primary concern appears to be the potential for catastrophic sinkhole development which might lead to failure of the on-site encapsulation alternatives.

As explained previously, the EPA is making this remedy proposal without sufficient site-specific data and without due consideration of available literature concerning engineering practices in karst terrains. Numerous publications are available concerning planning and design considerations in karst terrains. Methods have been developed for investigating the possibility of catastrophic sinkhole collapse and for developing sinkhole risk models. Engineering solutions for prevention of structural damage because of sinkhole collapse have been developed which, among others, include the use of geosynthetics, compaction grouting and specialized foundation design. In the event that a sinkhole collapse did occur, proven remediation techniques have been successfully used.

This alternative is similar to Alternative D with the addition of more stringent run-on/run-off controls and stabilization of affected soil prior to placing the affected soil under a composite cap. Although the potential for formation of a sinkhole which would cause failure of the cap is considered very unlikely to occur, additional engineering controls and solidification of the consolidated material are proposed in this alternative to further ensure that failure due to sinkhole formation will not occur and also to immobilize any dioxin (addressed in the Appendices).

The capped portion of affected material is relocated southeast of the original Alternative D location as shown in Figure 2. As a result, all affected soil is excavated, stabilized and consolidated under the composite cap which is designed per Section 6.1.3 of the

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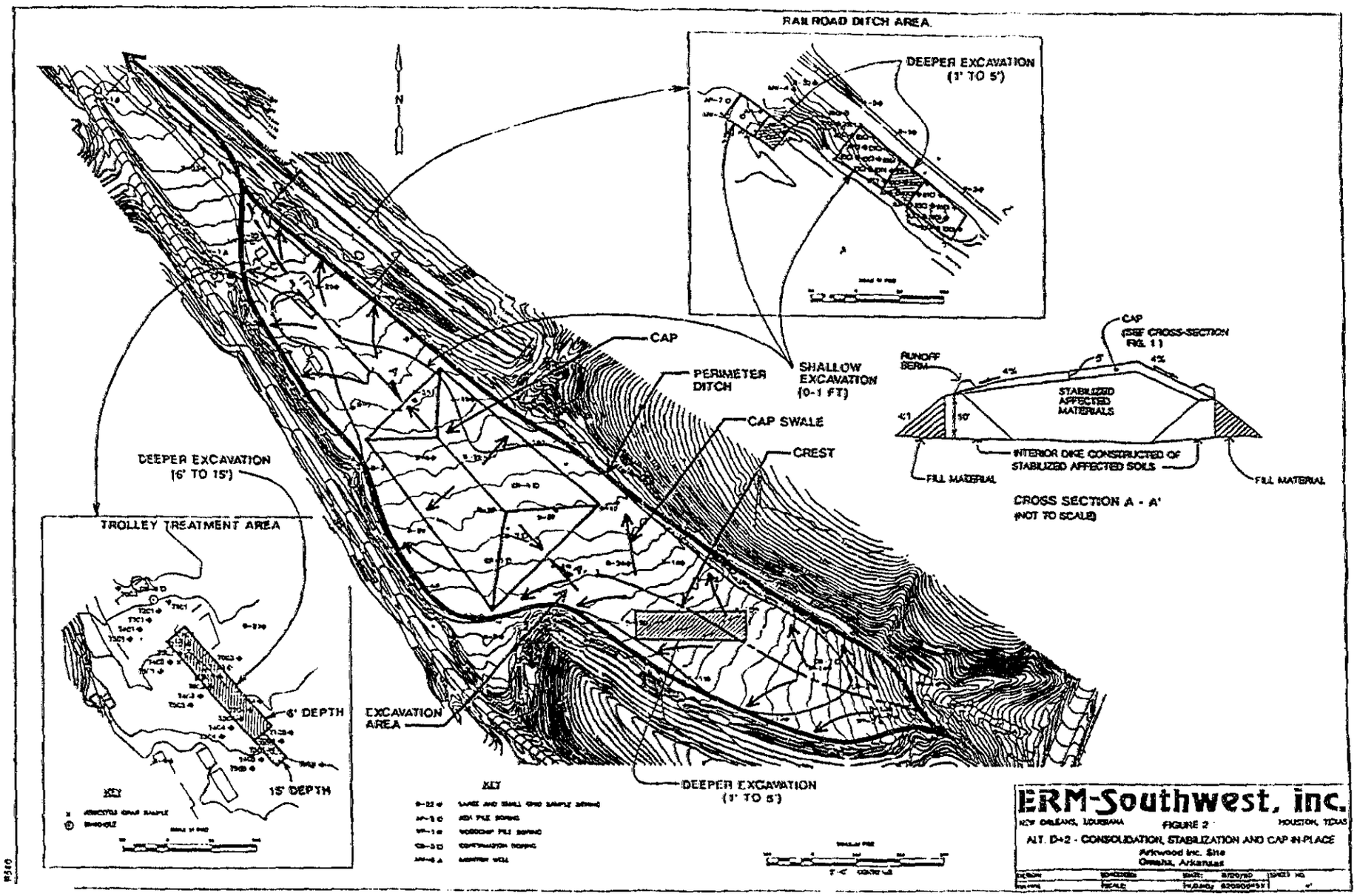
RAILROAD DITCH AREA
DEEPER EXCAVATION
(1' TO 5')
N
CAP
PERIMETER
DITCH
SHALLOW
EXCAVATION
(0-1 FT)
CAP SWALE
CREST
DEEPER EXCAVATION
(6' TO 15')
TROLLEY TREATMENT AREA
6' DEPTH
15' DEPTH
KEY
EXCAVATION
AREA
DEEPER EXCAVATION
(1' TO 5')
KEY
CAP
(SEE CROSS-SECTION
FIG. 1)
RUNOFF
BERM
4%
5'
4%
STABILIZED
AFFECTED
MATERIALS
4:1
10'
INTERIOR DIKE CONSTRUCTED OF
STABILIZED AFFECTED SOILS
FILL MATERIAL
FILL MATERIAL
CROSS SECTION A - A'
(NOT TO SCALE)
ERM-Southwest, inc.
NEW ORLEANS, LOUISIANA
FIGURE 2
HOUSTON, TEXAS
ALT. D+2 - CONSOLIDATION, STABILIZATION AND CAP IN-PLACE
Arkwood Inc. Site
Omaha, Arkansas

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FS Report. The remainder of the site is covered by a topsoil cap. Constituent migration, should it occur, is tracked by the monitoring program also described in FS Section 6.1.3. Sinkhole fluids and decontamination water are treated in an on-site wastewater treatment unit, described in FS Section 6.1.3, and discharged off site. Stabilization of the affected soil is achieved by the use of pozzolanic agents such as fly ash or kiln dust or with Portland cement mixed with the affected soil to produce a stable material which is strong, relatively impermeable, and resistant to physical breakdown. This process creates approximately a 24,000-cubic yard mass of stabilized material to structurally bridge any foundation settlement that may occur, such as from a sinkhole formation, in the unlikely event one should form. The stabilized soil will have a minimum compressive strength of 400 psi after 28 days of curing and will be similar in characteristics to a weak concrete. The Treatability Study Report indicates the use of silicate or lime additives will immobilize the high molecular weight organic compounds, produce the least pentachlorophenol (PCP) mobilization and yield the highest strength mixture. A pilot study to verify the appropriate stabilization process, quantity of additives required and that the mobilization of PCP can be minimized would be conducted during the Remedial Design.

The stormwater control system is shown in Figure 2 and consists of three major components: 1) a method to prevent stormwater from running onto the site, 2) a method to provide rapid removal of water falling on the site and 3) treatment of surficial areas prone to stormwater infiltration. Perimeter ditches are constructed to collect water running on the site and runoff from the cap. These ditches are designed with stone rip rap or concrete lining to accommodate high velocities. Drop structures, energy dissipators and collection boxes are required and will be located during the final design phase. Site runoff is controlled by providing a center crest with a 2 to 3% side slope and a series of swales designed to reduce the time of concentration for overland flow. The existing sinkhole and other near-surface features identified as being prone to stormwater infiltration are capped and graded in such a way as to divert stormwater around them.

Design Basis

See Section 6.1.3 of the FS report for the descriptions of fencing, decontamination and removal of existing structures, excavation and incineration of sludges, wastewater treatment, ground water monitoring and topsoil cap. Table 1 presents a detailed cost estimate for Alternative D+2.

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TABLE 1

Alternative D+2

Incinerate Sludges/Consolidate and Stabilize Soils/
Cap-in-Place Affected Soil and Provide
Stormwater Controls

Arkwood Inc. Site
Omaha, Arkansas

	Quantity	Units	Unit Cost	Cost[a]	Notes
CAPITAL & OPERATING COSTS					
Common Items				$310,000	See Table B-1B
Remove and Incinerate Sludges				837,000	See Table B-2
Line Perimeter Ditches/Rip-Rap	5,000	lf	25	125,000	
Dike Around Consolidation Area	1,530	lf	71	109,000	10' Height
Stabilize Affected Soils	20,400	cy	130	2,652,000	
Consolidate Affected Soils	23,500	cy	4.50	115,000	
RCRA Cap Over Consolidation Area	136,000	sf	3.00	409,000	Cap Shown in Fig. 6-7A
Topsoil Cap Over Remainder of Site	517,100	sf	0.74	383,000	
Site Facilities & Utilities - Capital				77,000	See Table B-1B
Site Facilities & Utilities - Operating	1-1/4	yr	183,000	229,000	See Table B-1B
Subtotal				$5,250,000	Rounded to ten thousands
Contractor Overhead, Profit & Bonds				x 1.20	
Contingency				x 1.25	
Engineering & Construction Surveillance				x 1.20	
				$9,400,000	Rounded
ESTIMATED CAPITAL & OPERATING COST					
POST CLOSURE CARE COSTS					
Maintenance	15	ac/yr	150	2,300	Rounded to hundreds
ce Maintenance	5,000	lf/yr	0.40	2,000	
Annual Subtotal				4,300	
Net Present Value of Annual Subtotal [b]				66,000	
Ground Water Monitoring (Net Present Value) [b,c]				194,000	
				260,000	
Contingency				x 1.25	
Engineering & Construction Surveillance				x 1.20	
NET PRESENT VALUE POST-CLOSURE CARE COST [b]				$390,000	
ESTIMATED ALTERNATIVE COST (NET PRESENT VALUE)[d]				$9,800,000	

NOTES.
[a] Costs are mid-1989
[b] Assumes an interest (discount) rate of 5% (net of inflation) and a 30 year post-closure care period.
[c] Monitoring frequency and parameters are specified in Section 6.
[d] The sum of capital and operating costs and the net present value of the the post-closure care costs.

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General:

Volume excavated	20,400 c.y.
Volume capped	25,500 c.y.
Area of cap	136,000 s.f.
Construction Period	1½ Year

Stabilized Material:

UCC Strength	> 15 psi @ 3d >300 psi @ 7d >400 psi @ 28d
Permeability	10^{-5} cm/sec. or less
Free liquid release	Minimal
Volume Increase	15 to 25%
Stabiliz tion Process	Silicate Additive or Lime Additive

While not warranted in our view, Alternative D+2 provides two advantages over the original Alternative D. First, an engineered system to control runoff from the entire site and surrounding area will reduce the already small potential for sinkhole development. Second, solidification of the affected soil placed under the composite cap will provide structural strength to withstand collapse should a sinkhole develop. Solidification would also provide additional assurance that dioxin and other constituents would be immobilized should the integrity of the encapsulated material be temporarily breached. This would eliminate any possibility of transport of constituents to the ground water system.

Solidification and cap-in-place has been used in numerous Superfund site remediations. In EPA's recent survey on technology trends in treatment RODs between 1987 and 1989, 45 sites, 22.5% of the total sites in the study, used solidification/stabilization as a means of site remediation. Examples of RODs containing solidification/ stabilization include Selma Pressure Treating Company in Selma, California; Love Canal - 93rd Street School, Niagara Falls, New York; and Diamond Alkali - 80 Lister Avenue, Newark, New Jersey.

Alternative D+2 is protective of human health and the environment and provides both short- and long-term effectiveness. It reduces toxicity by destruction of 40% of the contaminant mass and mobility by encapsulation and would be easy to implement using existing technology. It would also be much more cost-effective than the EPA's preferred soils remedy, providing the same overall reduction in risk for significantly less cost.

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IV. GENERAL ASSESSMENT OF EPA'S PREFERRED SOILS REMEDY

A General Considerations

Alternative H is critically defective in terms of two of the "primary balancing criteria" used to weigh major trade-offs among feasible alternatives -- Implementability and Cost-Effectiveness. We believe that EPA will find that Alternative H also lacks community acceptance for the reasons discussed below. Alternative H has an excessive cost with little or no increase in protection of human health and the environment, and is complex to operate and, we believe, will lack community acceptance.

B. Implementability

The EPA concedes that incineration is a complex alternative to implement (Proposed Plan, page 8). The construction of an incinerator is a formidable task that presents considerable engineering difficulties. It entails the careful assembly of many intricate parts that demand skilled construction engineers. Injuries and even deaths in the operation of incinerators have been recorded. Incineration requires highly trained specialists, who are often not readily available, for operation and maintenance. A trial burn would be required before beginning operations, and constant analytical work and monitoring would have to be implemented.

Incineration also presents two additional difficulties from the standpoint of operating the scrubber system and handling of materials. The scrubber system is required to prevent stack gas pollution. It requires a source of water and it creates wastewater. Additionally, the ash would need to be backfilled and covered with a topsoil cap.

Material handling prerequisites prior to feeding the incinerator will require storage systems. Control of fugitive dust emissions will be necessary. Often grinding is necessary prior to feeding and this may also result in dust generation.

While there are several basic types of incinerators, each one has its own set of inherent difficulties. The rotary kiln incinerators are large, expensive, and may require a secondary combustion chamber. The fluidized bed incinerator presents costly obstacles to the easy removal of ash, and it can have extensive refractory problems. The multiple hearth incinerator has a long residence time and similarly extensive refractory problems. Its slow temperature response also makes this type of incinerator large and expensive. The infrared incinerator is mechanically complex and presents problems in the feeding mechanism.

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To operate efficiently and provide uniform treatment of contaminated materials requires pre-treatment or sizing of the feed material. Sizing of the feed materials for Arkwood can be accomplished by use of the sieve and wash process determined to be feasible in the Arkwood Treatability Study. A homogeneous feed material allows the combustion chamber to operate efficiently and assures uniform heat treatment or destruction of the particles of affected soils.

C. Cost-Effectiveness

The purpose of remediating a site is to provide adequate protection to public health and the environment from the constituents present at the site. While such protection is a priority, the National Contingency Plan requires that cost be considered and compared with the benefit that will result from implementing a remedy. A reasonable level of protection implies that the protection afforded the public and the environment is acceptable according to the prevailing norms of risk analysis. Any protection beyond this reasonable level does not endow significant benefits to the public and the environment; diminishing returns result. Any expenditure of resources to achieve this greater degree of protection is unjustified. Therefore, cost-effectiveness is required to be considered in site remedy selection. Any remedy that has an excessive cost without a corresponding increase in overall protection of public health and the environment is not cost-effective.

In the case of the Arkwood, Inc. site, the proposed incineration remedy is the most costly of all feasible remedies for the site. Furthermore, it does not provide for a significantly greater degree of protection than other, less costly remedies. The FS has demonstrated that several other alternatives involving partial incineration, consolidation and capping would provide full protection to public health and the environment for substantially less cost. This protection is measured by carcinogenic risk (for public health) and by predicted levels of pentachlorophenol in ground water (for the environment). An additional parameter is the level of residual dioxin in soil that is allowed to remain. While incineration provides for these three clean-up parameters, other feasible alternatives do so at substantially less cost.

D. Community Acceptance

Community acceptance is a critical part of the evaluation of incineration as a remedial alternative. Historically, incineration has encountered substantial local resistance. Such opposition is usually based on the health risk that incinerators are perceived to present because of their emissions of combusted materials into the ambient atmosphere. Furthermore, there is a misplaced distrust of

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government and industry on the part of the public; this misperception leads to the belief that the complex technology characteristic of incinerators would not be operated properly. The public would moreover be fearful of additional dioxin production due to the high-temperature combustion of pentachlorophenol. These concerns are not technically sound but, nonetheless, local opposition has often resulted in either delays in construction or in cancellation of the entire remediation option.

At the Arkwood, Inc. site, a school is located on a hill 1/4 mile from the site; this arrangement could result in the top of the incinerator stack being almost level with the school grounds. It is possible, therefore, that the public might perceive a health risk to school children as a result of inhalation exposure to stack emissions. This fear would be heightened when the emissions are visible. In addition, the site is situated in a valley less than a mile from the town of Omaha. Gaseous emissions could collect in the valley and perceptively deteriorate the ambient air quality of an area much greater than the site itself. If the scrubber system necessary to the incinerator were to go down, there could be increased emission of noxious products, such as hydrochloric acid and NO_x products, which would deteriorate the atmospheric ozone. Malfunctioning of the scrubber system could occur as a result of operator errors, equipment malfunction, or power outage. While it is our opinion that these risks can be adequately addressed, community acceptance is often based on misperceptions.

Another factor influencing community acceptance is the effect an incinerator may have upon the aesthetics of the region. The Arkwood, Inc. site area is very scenic, and the presence of an incinerator could detract from the scenic value of that part of the Ozarks. Community objections to incineration based on these and other factors could result in delays in construction and in remediation of the site.

E. Summary

In summary, Alternative H has an excessive cost without a corresponding increase in overall protection of public health and the environment, and when compared with soil washing/in situ vitrification (see Section VI-C.) is less attractive in terms of the other four primary remedial evaluation criteria.

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V. SIEVE AND WASH

ERM-Southwest does not believe that treatment of the affected soil is warranted; however, an essential preliminary step for any remedial alternative in which affected soils are to be treated, is sieve and wash. This process, which removes rock material that is not affected above the action level, must be an integral part of any treatment scheme no matter what the treatment process chosen, be it in situ vitrification, slurry biodegradation/stabilization or incineration. The process significantly reduces the volume of materials that require treatment, at a relatively low cost, thus providing a more cost-effective remedial action without reducing the effectiveness of the remedial alternative.

A. Background

The surficial covering residuum at the Arkwood Inc. site ranges from a few feet to over 50 feet in thickness. This material is derived from the decomposition of the Boone Formation, a predominantly gray, finely crystalline crinoidal limestone with abundant thin layers of cherty limestone and interbedded gray dense chert. The residuum resulting from the weathering of this parent material is a red, very cherty clay. Visual observation of the residuum at the site indicates that the majority of the residuum is composed of chert rocks (chert is a microcrystalline siliceous rock) with lesser amounts of sands, silts, and red clays.

Since siliceous rocks do not readily sorb organic contaminants, it was postulated that the chert mass comprising the majority of the residuum did not contain significant concentrations of organic constituents of concern, PCP, polynuclear aromatic hydrocarbons (PNAs) or dioxins and dibenzofurans.

B. Particle Size Analysis of Residuum

A large composite sample of residuum was collected using a backhoe from several locations at the Arkwood Inc. site. This sample was well mixed, then drummed for use according to methodology described in the Treatability Study section of the FS. Particle size analysis of this residuum, as noted in Table 2, shows that approximately 70 percent of the bulk material is greater than 12 mesh (0.0555 inches) in size. This 70 percent mass is composed almost entirely of chert.

C. A Well Established System

Soil washing is a volume reduction process for treating excavated soil. Soil washing is based on the following premises: 1) contaminants tend to be concentrated in the fine size fraction of soil

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TABLE 2

Soil Sieving

Arkwood, Inc. Site
Omaha, Arkansas

Sieve Size	5/89 Total Soils Bulk Dry Sieve (% Finer) [a]	5/19/89 -12 mesh Analysis Wet Sieve (% Finer)	9/89 +1/4 Inch Bulk [a] Dry Sieve (% Finer) [a]	Combined Results (% Finer)	Grab Sample [b]
6 inch					100
inch					91.5
inch					87.3
3-1/2 inch					80.7
3 inch					74.1
2-1/2 inch					70.0
2 inch			87.9	92.7	65.8
1-1/2 inch					60.6
1-1/4 inch			71.1	82.5	
1 inch					51.8
3/4 inch			46.8	67.8	45.4
1/2 inch			27.8	56.2	39.1
3/8 inch					35.8
1/4 inch	39.4		0	39.4	
4 mesh (0.187")					30.4
10 mesh (0.0787")					28.0
12 mesh (0.0555")	16.7	100		16.7	
35 mesh (0.0197")		78.2		13.1	
40 mesh (0.0165")					26.2
48 mesh (0.0117")		71.9		12.0	
65 mesh (0.0083")		65.5		10.9	
100 mesh (0.0059")		61.1		10.2	25.8
200 mesh (0.0029")		53.1		8.9	25.4

Notes:

a] Separation into fractions for treatment tests.
b] Single bulk sample sieve analysis performed at Southwestern Laboratories, Inc. prior to Treatability Study.

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(silt, clay, and soil organic matter), and 2) contaminants associated with the coarse soil fraction (sand, gravel and cobbles) are primarily surficial. The objective of the process is to concentrate the contaminants into a smaller volume of material while producing a washed soil product meeting appropriate clean-up criteria.

Following debris removal, soil is mixed with water and subjected to various unit operations common to the mineral processing industry. Process steps can include mixing trommels, pug mills, vibrating screens, froth flotation cells, attrition scrubbing machines, hydrocylones, screw classifiers, and various dewatering operations. The core of the soil washing process is a multi-stage, counter-current, intensive scrubbing circuit with interstage classification. The scrubbing action disintegrates soil aggregates, freeing contaminated fine particles from the coarser sand, gravel and cobbles. In addition, surficial contamination is removed from the coarse fraction by the abrasive scouring action of the particles themselves. Contaminants may also be solubilized as dictated by solubility characteristics or partition coefficients.

These three mechanisms: - separation of contaminated fine particles, - scouring of coarse particle surfaces, and - dissolution of contaminants, each operate to varying degrees upon the soil and contaminant(s).

To improve the efficiency of soil washing, the process may be modified to include surfactants, detergents, chelating agents, pH adjustment or heat. In many cases, however, as was shown in the Arkwood, Inc. Site Treatability Study, water alone is sufficient to achieve the desired level of contaminant removal. A significant reduction in the volume of material requiring additional treatment or disposal is accomplished by separating the washed coarser soil components from the process water and contaminated fine particles.

The contaminated residual products can be treated by other methods. Process water is normally recycled following biological or physical treatment. Options for treating the contaminated fines can include in situ vitrification, biological treatment, incineration, or stabilization.

In the Arkwood, Inc. Site Treatability Study, washing studies on the coarse soil fraction were performed by combining an aliquot of a particular coarse soil fraction with tap water and tumbling this mixture in a standard laboratory ball mill without balls. The test simulated autogenous grinding in which the coarse soil material acted as the grinding media. This grinding action scours the surfaces of the coarse sand and pebble fraction, removing the surficial contamination. Three coarse soil size fractions were

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tested: -2" + 1-1/4"; -3/4" + 1/2"; and -1/4" + 12 mesh. Results indicated that:

- Washing was effective in removing both PCP and PNAs from the coarse soil to a level that would meet the expected cleanup criteria. Significant dissolution of PCP occurred in the wash water, but no detectable dissolution of the PNAs was observed. The dissolved PCP would be very amenable to biodegradation.

- Since the washing process is a grinding/attrition process, some of the coarse material is transformed into fines. The percent loss of coarse to fine increased with decreasing particle size. Thus the effectiveness of soil washing decreased with decreasing particle size.

- The removal of PCP appeared to improve with increasing particle size.

- The use of surfactants did not improve the soil washing process, but a subsequent caustic leach of washed soil did reduce the level of PCP in that soil.

In testing samples from the Arkwood, Inc. site, three coarse sieved-and-washed samples of different fractions were analyzed for carcinogenic and non-carcinogenic PNAs. The results are as follows:

[mg/kg]	Sample No. 1 (-1/4"+12 mesh)	Sample No. 2 (-3/4"+1/2")	Sample No. 3 (-2+1 1/4")
Total Nc-PNAs[1]	75	15	9
Benzo(a)anthracene	8.66	1.31	0.41
Chrysene	13.60	1.29	0.77
Benzo(b)fluoranthene	12.69	ND[2]	0.37
Benzo(k)fluoranthene	13.46	ND	0.38
Benzo(ghi)perylene	1.24	ND	ND
Benzo(a)pyrene	2.68	ND	ND
Total Benzo(a)pyrene Equivalents	6.7	0.2	0.14

[1] Expressed as phenanthrene, fluoranthrene, and pyrene.
[2] ND = Not Detected

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Washing studies on the fraction of soil that was less than 12 mesh was conducted by combining soil and tap water at 50 percent solids and subjecting this mixture to intense agitation in a laboratory flotation cell. As in the tests with coarse soil material, the high-intensity attrition scrubbing achieved in this apparatus results in the scouring of the surfaces of the medium and fine sand size material (-12 mesh + 200 mesh) removing surficial contamination. Results indicated that:

- Approximately 50 percent of the medium and fine sand size fraction (-12 + 200 mesh material) fed to the soil washing test exits the washing process as -200 mesh material. The remaining washed sand fraction constitutes approximately 3.5 percent of the total soils.

- The addition of surfactants or increasing the pH of the wash water did not lead to any significant improvement in performance over tap water alone.

- Washing did reduce the levels of both PCP and PNAs in the washed sand fraction of the soil by 86 and 48 percent, respectively.

- The low recovery of washed sand relative to the complexity of the commercial sand washing flowsheet suggests that soil washing would not be an economical option for this fraction of the Arkwood soil. Rather, this material should proceed directly to treatment such as bioremediation, in situ vitrification or incineration.

A relevant reference for soil washing can be found in Valine, Stephen B., Chresand, Thomas J., and Chilcote, Dennis D., "Soil Washing System for Use at Wood Preserving Sites", Proceedings of the 1989 Air and Waste Management Association/EPA International Symposium on Hazardous Waste Treatment, Biosystems for Pollution Control, February 1989.

Soil washing has been commercialized and is now used routinely in Europe. In the United States, the EPA has contributed extensively to the development of this technology through work at the Edison facility. The soil washing equipment that was fabricated by the EPA is now in the process of being transferred to the public sector through the Federal Technology Transfer Act. Finally, soil washing is the technology of choice in several recently issued Records of Decision (RODs). In a recent review of RODs that were issued for FY 1987-89, fifteen of the RODs (7.5%) contained soil washing/flushing (A Comprehensive Analysis of Remedies Selected in the Superfund Program During FY '87, FY '88, and FY '89, U.S. Environmental Protection Agency, June 20, 1990). Examples of

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Superfund sites where RODs contain soil washing as part of the remedial action are the South Calvacade site in Houston, Texas and the Koppers Texarkana, Texas site. Both are located in EPA, Region 6.

Thus soil washing is an innovative, yet tested and accepted technology for site remediation.

D. Applicability of Soil Washing to the Arkwood, Inc. Site Residuum

During 1989, BioTrol, Inc. conducted a comprehensive study of the applicability of soil washing to soils from the Arkwood, Inc. site in Omaha, Arkansas. These soils contained PNAs, PCP, and chlorinated dioxins/furans. This study characterized the soil from the site and evaluated the applicability of soil washing to various soil size fractions. The results of this study, Volume II, Appendix C of the FS Report, indicated that soil washing, within the expected guidelines for cleanup of PNAs and PCP, could reduce the volume of soil that would require additional treatment by approximately 65%. Soil washing was effective in achieving EPA's proposed clean-up criteria for PCP on the coarse soil fraction (>12 mesh).

Constituents of concern are both eroded from the large particles nd desorbed during the sieve and wash process. The remaining .race concentrations are not readily available to leaching as reflected in the Treatability Study results which showed that neither increases in pH of the wash solution or the addition of surfactants increased the quantity of PCP that could be leached from the coarse fraction of the residuum. It is therefore, highly unlikely that leaching under natural processes such as rainfall infiltrating through washed coarse residuum, would result in mobilization of constituents of concern.

Therefore, soil washing provides a cost-effective and scientifically sound technology to reduce the volume of residuum requiring treatment. It does this by cleaning and recovering a coarse soil fraction that requires no further treatment. The benefits of reducing soil volumes before treatment are obvious, including shorter treatment time which would enhance community acceptance. Reduction in volume is, in and of itself a SARA requirement. While we recommend against a treatment technology being included in the selected soils remedy, if one is, it must include an initial sieve and wash step to reduce volume and eliminate the treatment of vast quantities of unaffected rocks.

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SECTION 6

VI. ALTERNATE TREATMENT OPTIONS

A. Soil Washing/On-Site Incineration

One method of treatment would be to provide on-site incineration after soil washing.

A description of soil washing is detailed in Part V, Sieve and Wash.

As pointed out in Secti n IV.A., General Assessment of EPA's Preferred Soil Remedy, absence of community acceptance of on-site incineration is a significant issue, but soil washing to reduce the quanti-y of material to be incinerated would reduce the length of time the incinerator would be in operation. This would presumably reduce some of the community's objection.

The combined use of soil washing and on-site incineration would be consistent with documentation presented in the FS Report. Alternative E and F include soil washing. Although the FS Report did not combine complimentary features of these two processes explicitly, one can intuitively conclude that the use of soil washing to reduce the volume of material to be incinerated is feasible. With soil washing, the volume of material to be incinerated would be reduced by two-thirds to approximately 7,000 cy. Not including this combination as an alternative was an oversight.

Costs for this Alternative are given in Table 3.

B. Soil Washing/Biological Treatment/Stabilization

1. Introduction

Another method of treatment would be biological treatment following soil washing. Stabilization would also be done as a final procedure. During 1989, BioTrol, Inc. conducted a comprehensive study of the applicability of both soil washing and biological degradation to soils from the Arkwood, Inc. site. These soils contained PNAs, PCP, and chlorinated dioxins/furans. This study characterized the soil from the site, evaluated the applicability of soil washing to various soil size fractions, and determined the applicability of biological degradation for both PCP and PNAs. The results of this study were presented in the FS Report and have been used, along with those from a separate study within the FS to assess stabilization of site soil, to develop an innovative remedial process for the Arkwood, Inc. site, which consists of soil washing (sieve and wash), biological treatment and solidification.

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TABLE 3

Sieve and Wash/Incinerate Sludges and Affected Soil
Fines On-Site

Arkwood, Inc. Site
Omaha, Arkansas

	Quantity	Units	Unit Cost	Cost [a]	Notes
CAPITAL & OPERATING COSTS:					
Common Items				$310,000	See Table B-1B
Excavate sludge	430	CY	$3.50	2,000	
Excavate affected soils	20,400	CY	$3.50	71,000	
Sieve and Wash Affected Soils				1,060,000	
Percent Soil Meeting Treatment Goals	66%				
Incinerate Fines On-Site	7,000	CY	$430	3,200,000	See Table B-7A
Remaining Washed Soil for Capping	13,400	CY	$1.50	20,000	
Site facilities & utilities - capital				77,000	See Table B-3B
Site facilities & utilities - operating	1	years	$183,000	183,000	See Table B-3B
Backfill mixed ash & incinerator scrubber blowdown sludge	6700	CY	$1.50	10,000	
Wastewater Treatment				500,000	Preliminary Estimate
Restore Disturbed Areas	15	ac	$19,000	285,000	
total				$5,700,000	Rounded to hundred thousands
Factor Overhead, Profit & Bonds			X	1.20	
Contingency			X	1.25	
Engineering & Construction Surveillance			X	1.20	
ESTIMATED CAPITAL & OPERATING COST				$10,300,000	Rounded
ESTIMATED POST-CLOSURE CARE COST				$0	
ESTIMATED ALTERNATIVE COST (NET PRESENT VALUE) [b]				$10,300,000	

NOTES

[a] Costs are mid-1989.

[b] The same as the capital and operating cost since there are no post-closure costs for this alternative.

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2. Treatability Study

The goal of the Treatability Study was to identify potential treatment options for the Arkwood, Inc. site. The treatability study performed by BioTrol focused on the use of soil washing and biodegradation. Both technologies appeared to be applicable to the particular soils from the Arkwood, Inc. site, and a combination of the two technologies emerged as the technically effective option. This combination would allow soil washing to clean and separate the coarse soil particulates that met the cleanup criteria from the finer soil fraction that did not met the cleanup standards. Soil washing, although not effective on the total mass of soil, could significantly reduce the volume of soil that would require biological treatment, thus reducing the cost of this subsequent step. The biologically treated soil did achieve the proposed cleanup criteria for PCP. Details of the test results can be found in the document entitled "Feasibility Study - Volume II, Appendix C, Treatability Study Report," ERM-Southwest, Inc., March 30, 1990.

a. Soil Washing

A description of soil washing is detailed in Part V.

b. Biological Treatment

The effectiveness of biodegradation in a slurry system was evaluated to assess the applicability of this technology to the Arkwood, Inc. site. Initial studies were conducted to determine the existence of specific degraders indigenous to the Arkwood soil. Degraders specific for PCP were observed. A series of preliminary dilution studies were also conducted which showed no inhibition in biological activity over the range of 5 to 30 percent solids in the slurry. Based on the expected thickening characteristics of the Arkwood clay material, 15 percent solids was chosen as the level to use in the biological treatability study.

The bioslurry testing was performed on 100-gram aliquots of Arkwood, Inc. site soil (-12 mesh) placed in flasks containing inoculated nutrient media. The use of sacrificial replicates was chosen for this study so the total contents of the flask could be sampled at each sampling period. The flasks were agitated continuously on a gyratory shaker to provide mixing and aeration of the slurry. At any given sampling time, the flask contents were filtered and both solids and filtrate were analyzed separately. Samples were taken at days 0, 7, 14, 28, 56, and 98. Results indicated that over 89 percent of the PCP and 90 percent of the indicator PNAs were degraded in the time frame of the study. The biological activity for PCP exhibited three distinct phases, lag, active, and plateau. Soil samples subjected to the full duration

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of biodegradation still had residual levels of contaminants, but those contaminants were nondetectable in TCLP extracts. This indicates that the remaining constitutes, though present were sorbed to the soil matrix in such a manner as to be unavailable to biodegradation and desorption by standard physical or chemical processes in the TCLP leaching procedure. Thus biodegradation was capable of eliminating the exchangeable or available fraction of contaminants in the soil. (See Arkwood, Inc. Site FS Study, Vol. II, Appendix C, Treatability Study Report, March 30, 1990.)

c. Conclusions from the Treatability Study

The Treatability Study covered a wide range of options for the Arkwood, Inc. site, and a number of conclusions were drawn from the results, including:

- The Arkwood, Inc. site soils contain a high percentage of cobbles and gravel-sized material. Soil washing does lead to some attrition of this coarse material, producing soil fines. Following washing, approximately 66 percent of the total soil mass remains as coarse soil and approximately 34 percent remains as soil fines, finer than approximately 12 mesh.

- In general, the concentration of PCP and PNAs increases with decreasing particle size.

- For the coarse soil fraction, the efficiency of soil washing appears to improve as particle size increases. The coarse soils could be washed to achieve the proposed 300 ppm PCP and any reasonable cleanup level for the carcinogenic PNAs. Washing the medium and fine sand size fraction is not practical due to the limited volume of this material. Additives such as surfactants or changes in pH did not improve the efficiency of soil washing.

- Leaching previously washed soil in a caustic solution at high pH further reduces the PCP levels in the soil.

- Slurry biological treatment was effective in reducing the concentrations of PCP and PNAs in the -12 mesh fraction of site soils. The concentrations of PCP and PNAs were reduced by approximately 85 and 80 percent, respectively. The batch laboratory studies required extended treatment times to achieve complete bioremediation. The presence of a lag phase indicates that the treatment time can be reduced by acclimation.

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- In a separate phase within the Treatability Study, stabilization was tested as a potential technology for the untreated Arkwood, Inc. site soils. Stabilization was not effective for the untreated soils because it resulted in increased PCP mobility in the affected soils. However, the soil washing and biological studies have shown that PCP would not be available for mobilization after treatment has occurred. While immobilization of the PNAs does occur with stabilized soil, negligible leaching of PNAs occurred with untreated soil.

3. Developing an Innovative Technology Design for Arkwood, Inc. Site

The purpose of innovative technology is to provide a cost-effective remedial solution for any given site under the appropriate regulatory constraints of ensuring protection of human health and the environment. More appropriately, innovative process design can be used in which traditional and innovative technologies are combined in an optimum arrangement to address site-specific conditions. This design is most effective when conventional, industrial equipment can be used in the process circuit; i.e., when tested equipment is utilized in a unique configuration to accomplish a specific goal.

The various treatability studies showed some successes and some failures. Notably, the treatability studies suggested that:

- Soil washing was effective in achieving a 300 ppm cleanup level for PCP on the coarse soil fraction (>12 mesh), but not on the fine soil fraction.

- Biological treatment was effective in treating the mobile or available fraction of PCP and PNAs in the -12 mesh soil fraction. The residual PCP and PNAs were not leachable by TCLP. However, biodegradation has not been shown to be effective on chlorinated dioxins or furans.

- Stabilization is effective in immobilizing dioxin. However, it was ineffective in immobilizing the PCP in the untreated fine fraction of site soils.

Based on the results of the treatability studies, an innovative design for the Arkwood, Inc. site would consist of combining three technologies:

- soil washing, to clean and recover a coarse soil fraction and thereby reduce the volume of soil that would require additional treatment;

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- o bioremediation, to reduce the toxicity of the fine soil fraction by degrading the bioavailable constituents of concern in that soil fraction; and

- o stabilization, to immobilize the constituents of concern that were either not available for biodegradation (tightly bound PCP and PNAs) or not biodegradable (dioxins and furans).

a. Soil Washing

Soil washing is an integral part of any bioremediation system. A detailed discussion of soil washing is provided in Section V.

b. Bioremediation

Biological treatment is a generic term denoting a number of processes that use microorganisms (principally bacteria) to degrade organic contaminants from polluted environments, including soil, water, ground water, sludge, etc. Microorganisms are particularly useful for environmental clean-up because they produce enzymes that catalyze the degradation of organic molecules. The microbes often use the carbon, nitrogen and energy from the destruction of pollutants for growth.

The objective of biological treatment is to provide the proper microbiological environment for natural biodegradation processes to occur. This usually consists of adding inorganic nutrients (principally nitrogen and phosphorus) and assuring the presence of appropriate levels of moisture and oxygen. Occasionally, it is beneficial to use laboratory-grown microbes with unusual biodegradative capabilities. Biological treatment often results in the complete destruction of pollutants to non-toxic mineral end products. Since the maintenance of microbes is not costly, the process compares well economically with other treatment techniques.

Biological treatment has garnered strong EPA support, primarily through the Risk Reduction Engineering Laboratory in Cincinnati, Ohio, the Microbial Ecology/Biotechnology Laboratory in Gulf Breeze, Florida, and the Groundwater Research Laboratory in Ada, Oklahoma. Bioremediation, both in land treatment and in situ applications, is used extensively in the United States today. BioTrol has completed two SITE demonstrations of bioremediation technology, one using an aqueous bioreactor to treat groundwater contaminated with PCP and PNAs, and a second utilizing a slurry bioreactor to treat soil fines from a soil washing demonstration. This proven remedial technology has also been recommended in several recently issued RODs including the Koppers Texarkana, Texas

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site, and the Calvacade site in Houston, Texas, both EPA Region 6 sites.

c. Stabilization

Stabilization is a treatment process that is designed to accomplish one or more of the following results:

- o Improve the handling and physical characteristics of the waste, as in the sorption of free liquids.
- o Decrease the surface area of the waste mass across which the transfer or loss of contaminants can occur.
- o Limit the solubility of any hazardous constituents of the waste such as by pH adjustment or sorption.
- o Change the chemical form of the hazardous constituents to render them innocuous compounds or make them less leachable.

Stabilization achieves these results by mixing the contaminated material with water, various additives and pozzolanic materials or cement. This process produces a monolithic block of treated waste with high structural integrity. Stabilization techniques limit the mobility of the contaminants, whether or not the physical characteristics of the materials are changed or improved. This is usually accomplished through the addition of materials to ensure that the hazardous constituents are maintained in their least mobile or least toxic form.

Stabilization processes have been used extensively in the United States to treat sludges. SITE demonstrations have been completed by both HAZCON (Douglassville Superfund site, Douglassville, PA) and Soliditech, Inc. (Imperial Oil Company/Champion Chemical Company Superfund site, Morganville, NJ). Solidification was used to remediate the Pepper Steel and Alloys Superfund site (Pb and PCBs) in Florida. In a recent Proposed Plan of Action, biological degradation followed by solidification has been proposed as the permanent treatment of choice for soils contaminated with PCP, PNAs and dioxins at the Texarkana Wood Preserving Company Superfund site in Texarkana, TX.

d. Conceptual Treatment Process

A conceptual treatment process for the Arkwood, Inc. site soils would include soil washing, biological treatment and stabilization as illustrated in Figure 3. This is in reality a double protection plan. After employing soil washing to remove soil that does not require treatment, the remaining soil would be treated biologically to remove the acute hazard associated with the readily leachable

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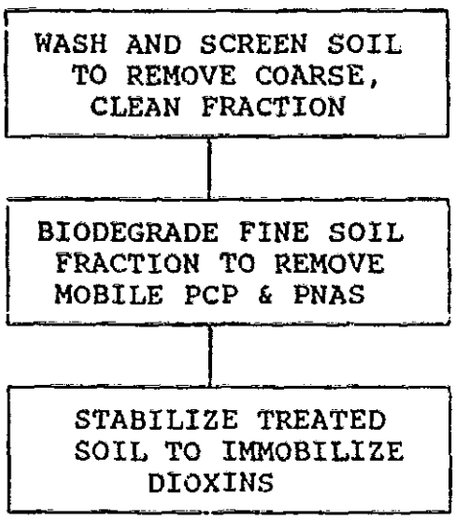
constituents of concern in this material. The residual contaminants of concern are bound very tightly to the soil matrix, since they are non-detectable by TCLP. This biologically treated and stabilized soil would then be subjected to solidification to eliminate any potential chronic hazard that may result from the residual contaminants of concern, including the dioxins and furans that are not amenable to biological oxidation.

FIGURE 3. CONCEPTUAL PROCESS TO TREAT ARKWOOD SOIL



flowsheet for such a treatment process for the Arkwood, Inc. site is shown in Figure 4. Note that this flowsheet is continuous as opposed to batch, and is designed to treat all affected materials on the site, including sludges. This flowsheet follows the conceptual outline in Figure 3, and is based on the results of the treatability studies that have been completed. Given this flowsheet, some additional optimization studies would be required at the bench level, to better define design criteria for the circuit. Following the bench-scale studies, a pilot circuit would be assembled and operated to develop the final design criteria for the commercial system.

The critical factor in the pilot operation would be the determination of the acclimatized, steady-state kinetics of biodegradation. Results from the bench studies showed that the appropriate indigenous organisms existed and that biodegradation was feasible. Although an estimated rate was calculated in the Feasibility Report, the treatability studies did not begin to develop the appropriate rate data for biodegradation, which can only be accomplished in a continuous system. Numerous studies have shown that continuous biological reactors, when operated under controlled

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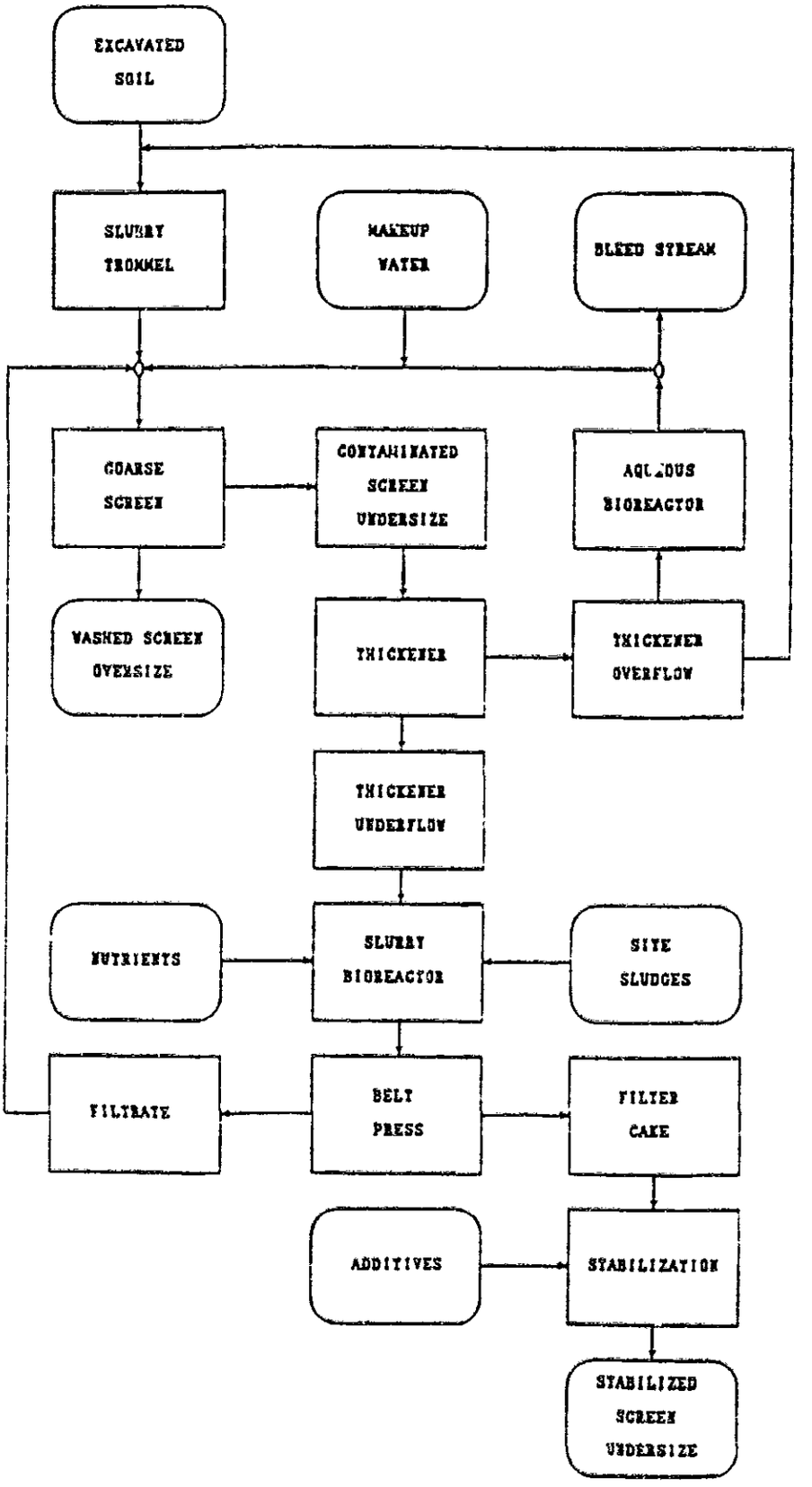
FIGURE 4 FLOWSHEET: PROPOSED TREATMENT PROCESS FOR THE ARENOOD SITE



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conditions, perform far better than would be expected from batch studies (Gunter Brox, EIMCO, personal communication). This forms the basis for the development of the EIMCO Biolift Reactor, which we would propose using for the actual remediation. Based on past experience, residence times on the order of a few days would be expected once steady-state conditions are achieved. This assumption would be verified in pilot studies prior to field remediation.

A treatment process flowsheet for the Arkwood, Inc. site is shown in Figure 5. The process equipment and recycle loops have been defined, and the preliminary control strategy has been established. This is a relatively simple, flexible process that would utilize conventional water treatment and mineral processing equipment. There are no mechanical or logistical issues that would prevent this process from effectively treating soil and sludges from the Arkwood, Inc. site. The final proof of the efficacy of such a process would come from a successful pilot testing program.

4. Conclusions

The results from a series of treatability studies indicated that soil washing, within the expected guidelines for cleanup of PNAs and PCP, could reduce the volume of soil that would require additional treatment by approximately 65 percent. The residual 35 percent of soil (the fine soil fraction) could be treated biologically, degrading 80-95 percent of the remaining PCP and reducing the levels of indicator PNAs to approximately 40 ppm. The contaminants that remained after biological processing were not detected by TCLP, indicating that biodegradation is also a stabilization technology relative to the remaining constituents of concern. Since dioxins and furans are not amenable to biological treatment, the residual soil could be stabilized following biological treatment to reduce mobility of all remaining hazardous constituents.

Based on the results of these studies, an innovative remedial process could be designed which incorporates soil washing, biodegradation, and stabilization. The integration of these three technologies results in a comprehensive, effective treatment process that reduces volume, toxicity and mobility of all constituents of concern at the Arkwood, Inc. site. This integrated process utilizes proven technology, employs standard industrial equipment, is protective of human health and the environment, and minimizes the impact of remediation on the local community.

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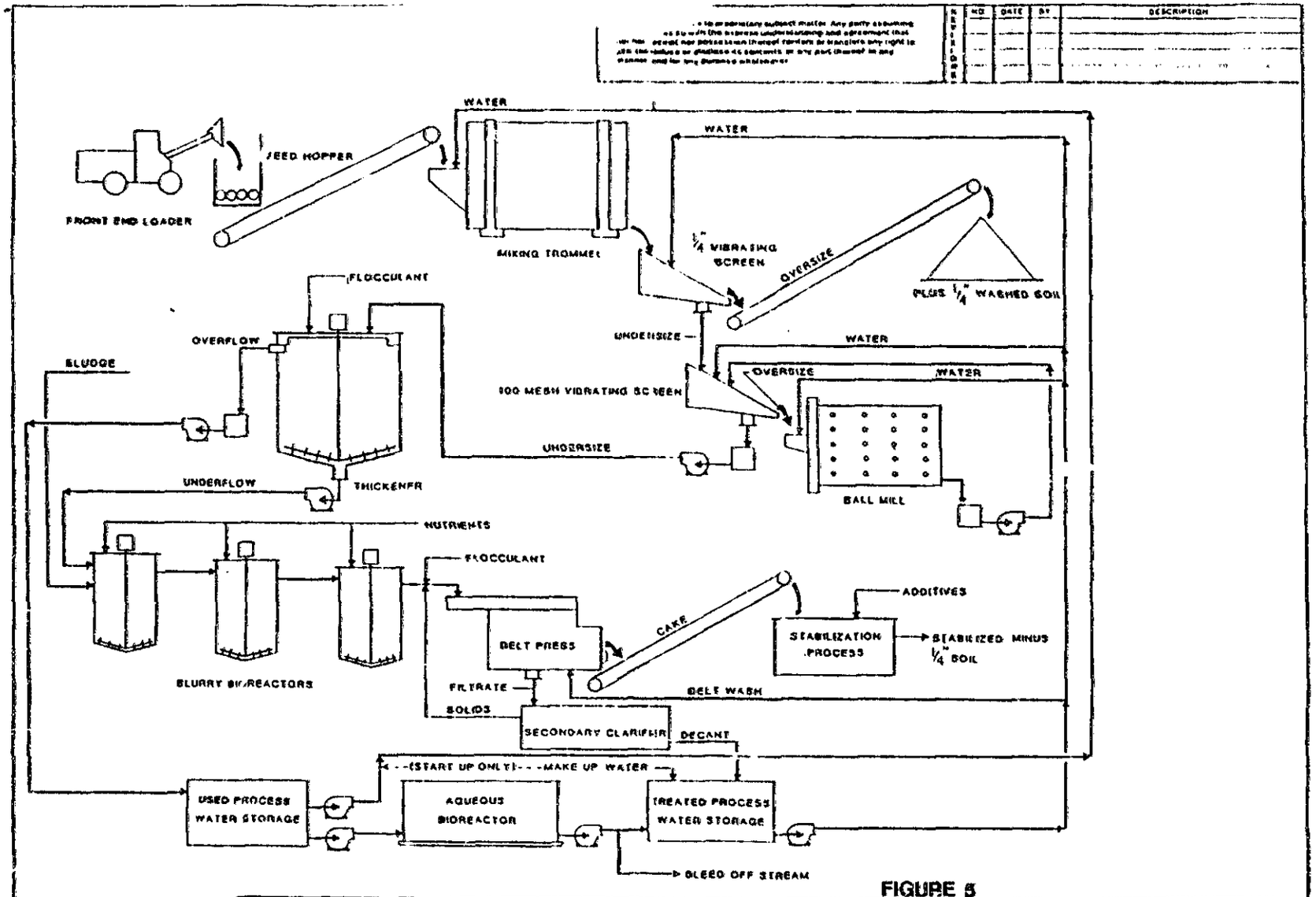

FIGURE 5

BIOTROL

51 PEAVEY ROAD CHASKA, MN 55318

PROPOSED TREATMENT PROCESS FLOWSHEET ARKWOOD SITE

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C. Feasibility Study of Soil Washing/In Situ Vitrification

1. Introduction

This section addresses the feasibility of using a combination of soil washing (sieve-and-wash) and in situ vitrification (ISV) to achieve desired treatment levels in affected soils and sludges. Taken collectively these two processes present an attractive alternative compared to Alternative H.

ISV is in situ electric melting of contaminated solids at high temperatures, typically in the range of 1600-2000° centigrade (C). Four electrodes are placed to the desired treatment depth. Since soil is typically not conductive enough in its natural state to allow process initiation, a graphite containing mixture is placed on the ground between the electrodes to provide a starter conducting path. Electrical current heats the starter path and the adjacent soil to temperatures above 1600°C.

Upon melting, soil becomes electrically conductive; thus the molten mass becomes the primary conductor and heat transfer medium allowing the melting process to continue downward and outward. The melt continues downward at a rate of 1 to 2 inches per hour. The ISV process results in a volume reduction ranging from 20% to more then 50%. The melted mass cools resulting in a glass monolith similar to volcanic glass.

Organics are destroyed by being broken down into their atomic constituents by the intense heat (pyrolysis). Volatile contaminants driven off as gases are collected by a negative pressure vent hood and routed through a treatment system where they are cooled, scrubbed, filtered and treated before release. Metals and nonvolatile constituents of inorganics are incorporated into the melt and become part of the glass which is highly resistant to leaching.

An ISV site is usually broken up into a "checker board" grid for treatment. A typical block in the proposed treatment grid would measure 25 feet by 15 feet by 15 feet deep.

Figure 6 shows a schematic of the proposed concept.

Several tests conducted by Geosafe, Inc., the vendor for this patented process, show that ISV is effective in the removal of a variety of organic and inorganic constituents. Table 4 provides a list of materials which have been processed in ISV tests. Table 5 shows typical removal efficiencies which can be expected for constituents found on this site.

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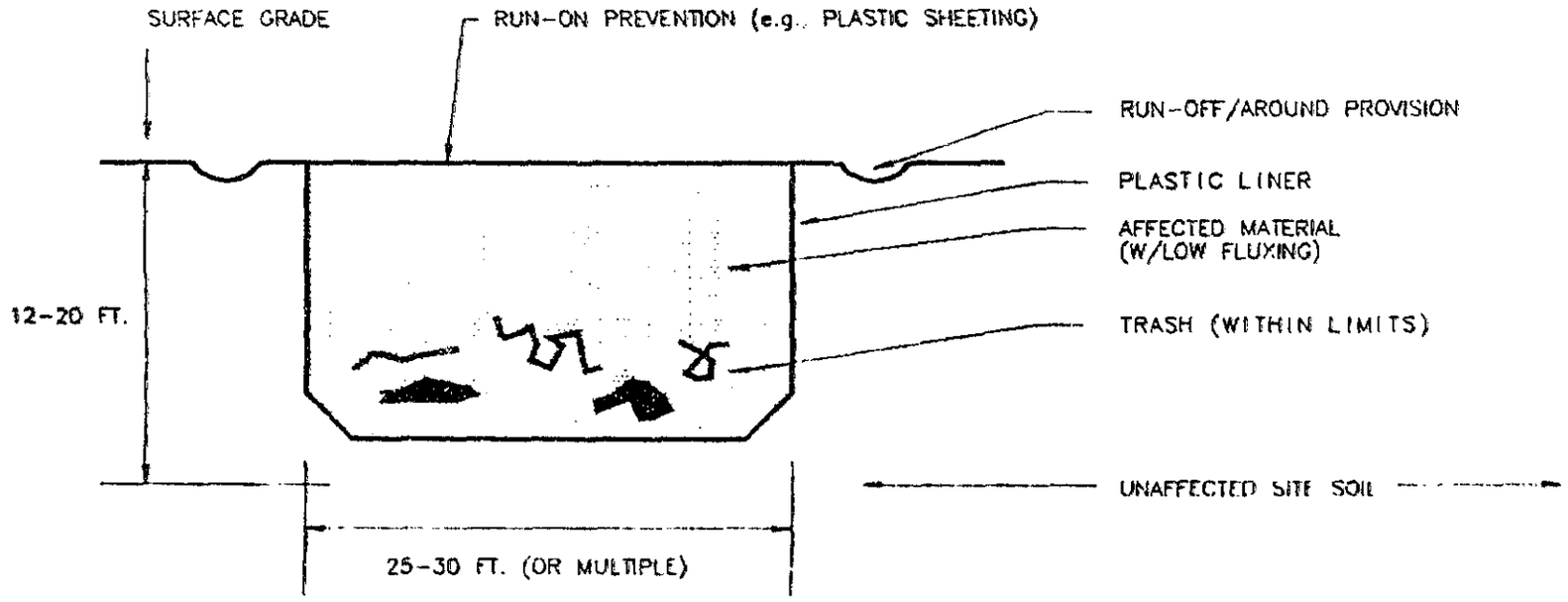

OTHER FEATURES:

1) TREATMENT TRENCH TO BE PLACED IN CLEAN SOIL, ABOVE GROUND WATER, AT CONVENIENT LOCATION.

2) LARGER ROCK (>12 MESH) REMOVED PRIOR TO STAGING BY SIEVE-AND-WASH PROCESS

3) SMALL FLUX ADDITION WILL "FOCUS" MELTING.

4) 30-40% VOLUME REDUCTION EXPECTED.

5) TOPSOIL OR TRENCH SPOIL BACKFILL (NO CAP NEEDED).

SOURCE: GEOSAFE CORPORATION

ERM-Southwest, inc.
NEW ORLEANS, LOUISIANA HOUSTON, TEXAS

DATE 09/11/90 | W.O.NO. 9214A002190

FIGURE 6
ISV STAGED IN SITU APPLICATION
CONCEPT FOR THE ARKWOOD INC. SITE

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TABLE 4

Waste Materials That Have Been Processed in ISV Tests

Heavy Metals	Liquid Organics	Solid Organics	Radioactive Materials
Lead	PCBs	Wood	Plutonium
Nickel	Dioxin	Buna Rubber	Amerecium
Cadmium	Trichlorethylene	PVC	Radium
Arsenic	Carbon Tetrachloride	Polyethylene	Uranium
Barium	Dichlorobenzene	Neoprene	Radon
Zinc	Benzene	Ion Exchange Resin	Cesium
Mercury	Methylene Chloride	Teflon	Ruthenium
Copper	Toluene	Paper	Cobalt
Aluminum	Ethylene Glycol	Cotton	Strontium
Iron	Methyl ethyl ketone	Polypropylene	
		DDT, DDD, DDE	

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TABLE 5

Typical Organic Destruction/Removal Efficiencies

Contaminant	Concentration (ppb)	Percent Destruction	Percent Removal (1)	Total DRE (%)
Organic Pesticides				
4,4' DDD, DDE, DDT	21-240,000	99.9 - 99.99	>99.9	>99.9999
Aldrin	113	>97	>99.9	>99.99
Chlordane	535,000	99.95	>99.9	>99.9999
Dieldrin	24,000	98-99.9	>99.9	>99.99
Heptachlor	61	98.7	>99.9	>99.99
Volatiles				
Fuel Oil	230-11,000	>99	>99.9	>99.999
MEK	6,000 (2)	>99	>99.9	>99.999
Toluene	203,000	99.996	>99.9	>99.99999
Trichloroethane	106,000	99.995	>99.9	>99.99999
Xylenes	3,533,000	99.998	>99.9	>99.99999
Semivolatiles				
Pentachlorophenol	>4,000,000	99.995	>99.9	>99.99999
Nonvolatiles				
Glycol	8,000 (3)	>98	>99.9	>99.99
PCBs	19,400,000	99.9 - 99.99	>99.9	>99.9999
Dioxins	>47,000	99.9 - 99.99	>99.9	>99.9999
Furans	>9,400	99.9 - 99.99	>99.9	>99.9999

NOTES

(1) This is the percent removed, from the off-gas, of the amount remaining after the percent destroyed; thus the percentages are additive for total DRE.
(2) 98% MEK in container, yielding 6,000 ppm in layer of container thickness.
(3) 50% ethylene glycol in container, yielding 8,000 ppm in layer of container thickness.

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2. Applicability of ISV to the Arkwood, Inc. Site

The first step in this alternative is soil staging by the sieve-and-wash process (see Section V). This process successfully separates the coarse portion of the residuum from the affected fine materials. A slurry containing this fine material is then dewatered and the filtrate returned to the washing process. The filter cake containing the concentrated constituents of concern (approximately 7,000 cy) is then transported to a trench and treated by in situ vitrification. Benefits of preceding ISV with soil washing are given in Table 6. (Soil washing is discussed in Section V.)

Following the soil washing process, affected material would be placed in a plastic lined trench located in an area which exhibits very low potential for formation of a sinkhole. Since excessive water is a deterrent to successful completion of an ISV project, necessary measures will be taken to preclude water from entering the treatment area. This will alleviate concerns about the karst geology causing problems during processing.

EPA has expressed a concern regarding sinkhole development on this site. ISV would provide an effective means of eliminating that issue because the residual monolith resulting from this process will be nonhazardous and structurally sound.

From a structural standpoint, after the material has been vitrified, the resulting monolith has a tensile and compressive strength typically about ten times that of unreinforced concrete. This results in a structural element which has the ability to span a large sinkhole in the unlikely event one should develop beneath the treated area. Additionally, after cooling, the ISV residual forms a structure physically and chemically similar to obsidian. This mass is so resistive to weathering it is expected to withstand exposure for geologic time periods of thousands to millions of years. If physical damage to the monolith did result in the production of fine particle sizes, the ISV residual has been shown to pass EP toxicity and TCLP testing criteria.

One of the more attractive features of ISV is the fact that all affected materials would be treated on-site. Excavation of affected materials is required for incineration or ISV processing; however, ISV offers the potential for treating some materials, namely that in the railroad ditch, in place without requiring any excavation. Additionally, materials generated as a by-product of the cleanup such as spent carbon, safety equipment, and so forth can be added to the trench where the affected soil is to be

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TABLE 6

Benefits of >12 Mesh Fraction Removal

1) Avoidance of melting large amount of uncontaminated material

2) Avoidance of need for increased levels of fluxant addition

3) Avoidance of possible interference by chert rock in melt dynamics

4) Shorter ISV processing time

5) Lower ISV costs

6) Greater public acceptance

7) Improved satisfaction of SARA mandate

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treated. Except for those materials associated with the last melt, all these items will either be consumed in the heat of the melting soil or will be encapsulated inside the monolith. The remaining small quantity of carbon can then be disposed of off-site.

With incineration, an ash will be generated which must be back-filled and covered with a topsoil cap. Conversely, the ISV process does not generate any additional materials. Once the soil melting period is complete and the mass is cooled, the process is finished and no further maintenance is required.

Another advantage to the use of ISV is that once the process is underway, treatment of the material is quite rapid. After the initial melt is begun on the surface, the heated material begins to move downward through the affected material processing it at a rate of approximately 1 to 2 inches per hour. At the planned trench depth of 15 ft, this would result in a processing time of 7 to 15 days for each zone of material. Total processing time would be on the order of 6 months to 1 year after startup. By contrast, treatment by on-site incineration would take as long as 2 years to complete.

Concern may be expressed that ISV is an innovative technology. While the process is certainly innovative, it is not unproven. As for being innovative, SARA charges EPA to favor such technologies. ISV meets the criteria for innovative technologies listed in CERCLA guidance, including:

- o Better treatment implementability. There would be less movement of equipment to the site and less installation/construction required. This is reflected in the mobilization costs for ISV, which are less than half those for incineration.

- o Fewer potentially adverse impacts. Tighter control on air emissions is attained with the ISV emissions hood than is attained with standard incinerator air pollution control devices (APCD).

- o Lower costs for similar levels of performance. Estimated total project costs for ISV are two-thirds of those for EPA's preferred soils remedy, with equal destruction and removal efficiencies (DRE) achieved. The main reasons for this are the higher mobilization, construction and decontamination/demobilization costs associated with incineration because of the type and amount of equipment required (i.e., kiln or primary thermal chamber, secondary chamber for combustion of off-gases, treated off-gas stack).

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3. Advantages of ISV

The ISV process has numerous advantages over Alternative H. These include:

- o Lower cost
- o Higher site-wide organic DRE through recycling of captured off-gas contaminants
- o Lesser potential for formation of dioxins/furans through oxidation of PCP
- o Significant (25-40%) residual product volume reduction (n.b., incineration of clay can result in volume increase)
- o Unequalled residual product from structural, longevity, leaching, and biotoxicity viewpoints
- o Significantly better off-gas treatment safety (approximately 1/7 the off-gas volume to be cleaned up)
- o Significantly better energy consumption (1/2 to 1/3 the thermal equivalent of incineration)
- o Greater overall safety
- o Greater community acceptance expected

4. ISV Precedents

Selection of ISV as the preferred alternative is not without precedent. At this time, ISV is currently being considered for use at several Superfund and RCRA Corrective Action sites. In fact, EPA has selected the process for remediation of one Superfund Removal Program site and two Remedial Program Sites. Treatment requirements at these sites represent a variety of constituents including PCP, dioxin, pesticides-herbicides, PCB's, lead and so forth. At one of these sites, Crystal Chemical Company in Houston, ISV emerged as EPA's preferred remedy to treat waste containing arsenic and low levels of organics. EPA is in the final decision process on the Crystal Chemical site.

Sites where ISV is the chosen or preferred remedy include:

1. Parsons Chemical/ETM Enterprises Superfund Site
   Grand Ledge, Michigan
   Region V Superfund Removal Program/EPA Lead
   EPA Funded
   No ROD, but removal program equivalent
   Pesticides/Dioxin/Mercury
   2-4,000 Tons
   EPA expects to issue contract by 9/30/90

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2. Northwest Transformer Superfund Site
Everson, Washington
Region X Superfund Remedial Program/EPA Lead
PRP Funded
ROD, Contingent upon successful T-Testing
Treatability test complete, awaiting analytical results
PCBs only
1,500-2,500 Tons
Consent order requires rapid schedule after successful T-Testing

3. Ionia City Landfill Superfund Site
Ionia, Michigan
Region V Superfund Remedial program/EPA Lead
PRP Funded
ROD, Contingent upon successful T-Testing
Mixture organics/inorganics, some rubble/debris
6,000 Tons
PRP work planning now in progress

4. CRAB Orchard Wildlife Refuge Superfund Site
Carbondale, Illinois
Region V Superfund Remedial Program/EPA Lead
PRP Funded
ROD, Contingent upon successful T-Testing
PCBs/Lead
40,000 Tons
PRPs pursuing T-Test planning

5. Transformer Service Facility Superfund Site
Spokane, Washington
Region X Superfund Remedial Program/State Lead
PRP Funded
Consent Decree Between state and PRP
PCBs only (to 10,000 PPM)
2,700 Tons
Final contract negotiations underway
This will be demonstration for National TSCA Permit

6. Shattuck Chemical Superfund Site (One of Denver Radium Sites)
Denver, Colorado
Region VIII Superfund Remedial Program/State lead
EPA Funded
Special Large-Scale demonstration project for EPA/HQ
Radium/Radon/Organics
900 Tons
Awaiting review of T-Testing Report which was just issued.

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7. M-1 Holding Ponds Site
Rocky Mountain Arsenal/Commerce City, Colorado
DOD IRM Site/Corps of Engineers managed
DOD Funded
DOD Equivalent of ROD
Arsenic/Mercury/Various Pesticides-Herbicides
15,000 Tons
30% Remedial Design just completed

8. Site 10
Arnold Air Force Base, Tennessee
Region IV RCRA Corrective Action Site
DOD IRM Site/Hazwrap Manage'
DOD Funded
DOD Equivalent of ROD
Mixture organics/inorganics (Old Fire Training Pit)
8,000 Tons
Approved through systems command (Top Level); Awaiting Funds

9. Crystal Chemical Superfund Site
Houston, Texas
Region VI Superfund Remedial Program
PRP Funded
ISV Preferred Remedy/ROD expected to issue by 9/30/90
Arsenic/Low levels of organics
17,000 Tons
PRP resisting EPA's selection as preferred remedy; EPA in final decision process now.

10. Anderson Development Superfund Site
Adrian, Michigan
Region V Superfund Remedial Program
EPA Funded
ISV Preferred Remedy/ROD expected to issue by 9/30/90
PCBs/Metals
4,000 Tons
PRP Vigorously resisting EPA's selection as preferred remedy; EPA in final decision process now.

11. Wasatch Chemical Superfund Site
Salt Lake City, Utah
Region VIII, Superfund Remedial Program
PRP Funded
Approved FS Recommends ISV
PCP/Dioxin
8,000 Tons
PRP/EPA/State have consensus on ISV as preferred remedy; preparations for public hearing underway.

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5. Implementability of ISV

The ISV vendor, Geosafe, Inc., has the personnel and mobile treatment equipment available to implement the ISV treatment process, and has indicated that the process is technically applicable to affected soils and sludges at the Arkwood, Inc. site.

Regarding availability of services, Geosafe acquired one ISV machine operational during 1989. Additional machines will be procured as the expected market for ISV materializes. Geosafe is able to procure additional machines within four to six months, which is significantly less than the time typically required to perform treatability testing, permitting/compliance, and remedial design efforts prior to on-site operation. Geosafe is willing to consider purchase of new machines for specific projects if the client has a schedule need that cannot otherwise be met by existing equipment. Geosafe will make schedule commitments for its machines given a corresponding commitment by the client.

Geosafe does not anticipate difficulty acquiring and maintaining field operations crews for its ISV systems. Geosafe conducts its own specialized ISV training program at its training center in Richland, Washington.

Because Geosafe holds worldwide exclusive rights to the ISV technology in the field of hazardous waste remediation, it is not possible for a client to obtain competitive bids for the ISV portion of a project. However, competitive bidding is possible on the related site preparation and general contractor support work.

6. Comparison of Soil Washing/ISV to Alternative H

ISV appears to be a cost effective remedy when compared to Alternative H, on-site incineration. The main reasons for this are:

- The shorter processing period reduces operating costs.
- Specialized equipment required for ISV is trailer mounted and can be quickly and easily moved onto the site.
- Energy consumption is lower than required for incineration.

Moreover, the ISV process appears to have the potential for better acceptance by the community. Even though the process has had limited commercial application to date, there has been significant media inquiry and reporting on it, and it has been received positively a number of times in various public hearings.

ISV has several advantages that relate to community acceptance, including:

- o the unequalled quality of its residual product,
- o a non-threatening appearing (and sounding) on-site equipment system,
- o the absence of any prior adverse record of misapplication or failure to perform, and
- o a very credible scientific basis and state of development by the U.S. DOE and Battelle Memorial Institute.

There is a public familiarity with glass and natural obsidian, and a recognition that they are essentially not degraded by the environment; this public perception together with technical data on the ISV residual product supports the public recognition that contaminants are permanently and harmlessly bound in the ISV residual product.

Additionally, it is noted that neither ISV nor Geosafe has any negative performance record that can adversely affect community acceptance. Incineration has public image difficulties (e.g., a smoke-stack image).

The extensive development program leading to the commercialization of ISV technology, and the excellent technical reputation of the developing organizations (e.g., U.S. DOE, Battelle), also provide a significant degree of credibility to the technology's acceptance. The technology has undergone significantly more development and test work than most other new technologies being introduced to the impending remediation marketplace.

Table 7 provides a comparison of ISV and incineration.

For comparison with Alternative H, a sieve and wash/in situ vitrification alternative has been written in the FS Report format and is included as Appendix F.

7. Recent Improvements to ISV

Although ISV was considered at the time of pre-screening of alternatives approximately two years ago, it was discarded as a potential remedial technology because of perceived technical difficulties and because it was not viewed as a commercially available technology at that time. However, in the intervening two years, significant developmental activities in the area of ISV technology have been carried out, and initial problems have been

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TABLE 7

Comparison of ISV to Incineration

Remediation Criteria	ISV	Incineration
1. Permanence/estimated life of remediation	Geologic time period	Permanent for organics, not applicable to inorganics
2. Reduction of toxicity	Organics destroyed beyond EPA DRE requirements	Organics destroyed beyond DRE requirements
3. Reduction of mobility	Inorganics permanently "immobilized" in residual	None for inorganics
4. Reduction of contaminated volume	25%-30% reduction minimum for most soils	Very small, to extent of organic volume (e.g., 1-5%)
5. Performance on-site	Yes - mobile, on-site	Yes, mobile, on-site
6. Performance in situ	Yes, with or without staging	No, not possible
7. Reduction of long-term liability	Maximum possible benefit	Good for organics, not applicable for inorganics
8. Cost effectiveness	Equal or lower cost	Equal or higher cost
9. Other operations required	Excavation and staging of sand/fines required	Excavation, pretreatment, treatment of large volumes of combustion gases, complex interrelated components, high probability of frequent downtime
10. Safety	Lowest possible occupational exposure, zero transport risk, inherent in situ safety benefits	Higher occupational exposure risk, zero transport risk
11. Community acceptance	Very good	Poor image

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solved. The erosion of electrodes which significantly limited the depth of potential melts has been solved with the development of electrodes that move down as the melt progresses. Off-gas treatment systems have been improved as have the efficiency and size of the hood used to cover the melt. Numerous laboratory, engineering and pilot scale melts have been carried out. The results of these tests prove ISV to be a viable technology for hazardous material destruction and immobilization. Also, in the interim, the technology was commercialized. Batelle has advanced the commercial credibility of the process so that ISV is now a viable remedial option being selected, or proposed for selection, at numerous sites (see Section 4, above).

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SECTION 7

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VII. EVALUATION OF EPA'S CANCER RISK ASSESSMENT FOR THE ARKWOOD, INC. SITE

Dr. Jon Rauscher, Toxicologist for the Texas Remedy Section, EPA Region 6 authored the memorandum entitled, "Upper Bound Excess Lifetime Cancer Risk and Remediation Goals for the Arkwood Superfund Site," July 6, 1990. The memorandum consists of calculations of carcinogenic risk associated with exposure to dibenzodioxins. Although the memorandum employs some of the exposure parameters used in the August 30, 1989 "Endangerment Assessment, Arkwood, Inc. Site," the underlying methodology is not consistent with the risk assessment document that was approved as final by the EPA Region 6.

There are three categories within the Rauscher memorandum that ERM-Southwest considers inappropriate:

1. the derivation of dioxin concentrations;
2. the procedure for assessing total risk; and
3. the target level for carcinogenic risk.

A. Derivation of Dioxin Concentrations

The dioxin concentrations employed in the Rauscher memorandum are presumably the upper 95 percent confidence limit of the arithmetic mean. The arithmetic mean is not the mathematically proper value to use for this data base in that it results in an overly conservative value. As was demonstrated in the Arkwood Endangerment Assessment (Section 4.3, "Estimation of Concentration of Indicator Chemicals at Points of Exposure"), the Arkwood, Inc. site concentrations are log-normally distributed, not normally distributed. The most appropriate measure of the average value for a normal distribution is the arithmetic mean, but for a log-normal distribution, the correct value for the average is the geometric mean (R. O. Gilbert, Statistical Methods for Environmental Pollution Monitoring, Van Nostrand Reinhold, 1987, Ch. 12 and 13). The geometric mean has been employed by EPA Region 6, e.g., at the Chrome I and II Sites in Odessa, Texas. There are substantial differences between the upper 95 confidence limit, arithmetic mean, and geometric mean for a given data base. These differences can have a significant impact on the accuracy of risk assessment. For example, for the dioxin data at the Arkwood, Inc. site, we observe the following values:

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[ppb]	Geometric Mean	Arithmetic Mean	Upper 95 Percent Confidence Limit of Arithmetic Mean
RC Grid	1.8	36.5	62
TC Grid	1.5	6.2	10
B Grid	1.5	12.4	18

These data are calculated using the revised Toxicity Equivalent Factors of 1989 to estimate 2,3,7,8 - TCDD equivalents. The data indicate the substantial differences among these various values. The lowest of these is the geometric mean, yet it is the most mathematically correct procedure for determining representative concentrations for an environmental data base. Therefore, to use the other values in dioxin risk assessment is not only mathematically improper but leads to an overestimate of exposure and risk by up to an order of magnitude.

B. Procedure for Assessing Total Risk

The Rauscher memorandum calculates total cancer risk by simple summation of the chemical intake of each age group, and then by calculating the risk from that single value. This same method was utilized in the original draft of the Arkwood Endangerment Assessment, but Region 6 stated that the method was inappropriate and required the use of time-weighted-averaging (TWA) for each exposed age group (EPA Comments on Arkwood Endangerment Assessment from Garret Bondy to R. H. Fuller, ERM-Southwest, Inc., July 28, 1989). This revision was incorporated into subsequent drafts of the document. Although there is no significant difference between simple summation and TWA (with TWA providing a slightly lower estimate), the TWA method should be considered the preferred method. Furthermore, the memorandum demonstrates an inconsistency in approach to assessing risk at the Arkwood, Inc. site by EPA Region 6.

C. Target Carcinogenic Risk Level

The Rauscher memorandum suggests in its mathematical formula for calculating PAH remediation goals (page 4), but does not overtly state in the text, that a target cancer risk of 10^{-6} is the remediation goal for the Arkwood, Inc. site. This is inconsistent with the future industrial land use pattern proposed for the site. A risk level of 10^{-6} can be appropriate for a residential area, but this land use pattern is not expected to be applicable to the

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Arkwood, Inc. site. For an industrial area, a risk level of 10^{-5} or even 10^{-4} can be applied with adequate protection of human health and the environment. For example, at the Geneva Industries site in Houston, Texas, a PCB clean-up level of 100 ppm was recommended by the Record of Decision (September 1986); this level corresponded to a 10^{-4} cancer risk level as determined by the risk assessment for the site (IT Corp., November 1985).

While not adopted in the Proposed Plan, the formula in the Rauscher memorandum for the calculation of a PNA clean-up level at the Arkwood, Inc. site utilized a total exposure time of 30 years. This is in contrast to a period of 10 years stated by Region 6 to be applicable for industrial settings (Ruth Izraeli and Jon Rauscher, personal communication, April 23, 1990).

D. Conclusions

It can be concluded that the EPA risk assessment does not employ methodology consistent with the original, approved Arkwood Endangerment Assessment or even with Region 6's own policy. This risk assessment should therefore be disregarded in evaluating cancer risk at the Arkwood, Inc. site.

E. Supplemental Materials

After the Arkwood Feasibility Study and Endangerment Assessment had been approved by EPA Region 6, a new set of toxicity equivalent factors (TEFs) were introduced to the Arkwood, Inc. site, by the Agency that substantially changed several of the concentration of dioxin congeners at the site, especially the octa-chlorinated homologue. In view of the concerns raised by the changing dioxin values, several supplemental documents are hereby provided to assess the impact of the revised dioxin concentrations on the endangerment assessment and on remedy selection. The following documents are found in the Appendices:

- "Evaluation of the 1989 Endangerment Assessment for Arkwood, Inc. Site";
- "Review and Critique of the Scientific Basis of the TEF for OCDD".
- "Alternatives to Incineration for Soils Containing Greater than 20 ppb Dioxin";
- "Dioxin Clean-up Goals for Arkwood, Inc. Site"; and

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- "Calculation of Preliminary Health-Based Soil Cleanup Levels for Polynuclear Aromatic Hydrocarbons (PAHs) at the Arkwood Site in Omaha, Arkansas"

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SECTION 8

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VIII. ADMINISTRATIVE RECORD

A. Incomplete Record

In response to the EPA's invitation to the public to comment on the Public Record, MMI makes the following comments:

The Administrative Record Index, Draft Final, for the Arkwood, Inc. site (ARK 084930148) dated July 12, 1990 does not include many of the letters, reports and other documents that were submitted by MMI to the EPA or received by MMI from EPA, during the RI/FS. Several of these are significant in terms of documenting the RI/FS progress and procedures. MMI hereby requests that the following documents be included in the Administrative Record:

Miscellaneous Matters

1. Letter from Larry D. Wright to Bob Barker, 3/4/87: Necessity to key sampling events to hydrological monitoring data.

2. Letter from Bob Barker to Ruth Izraeli, 3/5/87: Revisions to Arkwood, Inc. Site Work Plan; i.e., revised monitor well construction details, revised Health and Safety Plan, revised QA/QC Plan and gaging station proposal.

3. Letter from Richard Fuller to Ruth Izraeli, 3/10/87; RMAL QA/QC procedures.

4. Letter from Richard Fuller to Ruth Izraeli, 3/16/87: Proposed geophysical investigation and qualifications of ecologists doing site ecological assessment.

5. Letter from Ruth Izraeli to Bob Barker, 4/6/87: Comments concerning revised work plan documents.

6. Letter from Richard Fuller to Ruth Izraeli, 4/20/87: Preliminary results of well and spring inventory and selection of wells and springs to be sampled.

7. Fax from Ruth Izraeli to Richard Fuller, 4/28/87: Preliminary acceptance of wells and springs to be sampled.

8. Letter from Richard Fuller to Ruth Izraeli, 5/1/87: Response to EPA comments on revised work plan documents.

9. Letter from Richard Fuller to Ruth Izraeli, 9/25/87: Response to EPA questions concerning need to coordinate

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spring and well sampling events with meteorological and hydrological investigations.

10. Letter from Richard Fuller to Ruth Izraeli, 10/23/87: Revisions to Arkwood, Inc. Site Work Plan.

11. Letter from Richard Fuller to Ruth Izraeli, 4/9/88: Response to Ruth's questions concerning samples to be collected for determining target parameters.

12. Letter from Steve Calhoun to Ruth Izraeli, 7/7/88: Proposal for conducting EM geophysical survey.

13. Letter (unsigned) from Ruth Izraeli to Bob Barker, 7/13/88: Additional compounds to be added to indicator list.

14. Letter from Richard Fuller to Ruth Israel, 9/23/88: Elimination of gauging station at southeast end of railroad tunnel.

15. Letter from Steve Calhoun and Richard Fuller to Ruth Izraeli, 9/27/88: Procedures for conducting Phase 1C soil sampling.

16. Letter from Steve Calhoun and Richard Fuller to Ruth Izraeli, 10/4/88: Procedures for use and documentation of high volume air samplers.

17. Letter from Stephen Doss to Richard Fuller, 10/6/88: Weston's comments to Phase 1C soil boring proposal.

18. Letter from Richard Fuller to Ruth Izraeli, 10/11/88: Reply to EPA technical comments on Phase 1C soil boring proposal.

19. Letter from Richard Fuller to Ruth Izraeli, 10/28/88: Discussion of problems related to high laboratory detection limits for PAH compounds.

20. EPA internal memorandum from Randall E. Brown to Larry Wright, 11/15/88: Disposal of Wood Treating Wastes from the Arkwood, Inc. Superfund Site.

21. Letter from Richard Fuller to Brent Truskowski, 12/27/88: Final Phase I monitor well installation procedures.

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22. Letter from Richard Fuller to Brent Truskowski, 1/4/89: Revisions to final Phase I monitor well installation procedures.

23. Letter from Richard Fuller to Brent Truskowski, 1/24/89: FS alternatives analysis; i.e., preliminary assembly of remedial alternatives.

24. Letter from Richard Fuller to Brent Truskowski, 2/6/89: Revisions to Treatability Study Work Plan.

25. Letter from Garrett Bondy to Richard Fuller, 2/26/89: Comments on Treatability Study Work Plan.

26. Letter from Brent Truskowski to Richard Fuller, 3/23/89: EPA approval of Soil Sampling Plan.

27. Letter from Sam Becker to Richard Fuller, 4/25/89: EPA's approval of Final Treatability Study Work Plan.

28. Letter from Garrett Bondy to Bob Barker, 6/22/89: EPA contractor comments and recommendations for use in developing Phase II Work Plan.

29. Letter from Bob Barker to Brent Truskowski, 7/17/89: MMI's decision not to proceed with interim removal action.

30. Letter from Richard Fuller to Brent Truskowski, 7/20/89: Final revisions to Phase I Remedial Investigation Activities Summary Report (response to second round of EPA comments).

31. Letter from Richard Fuller to Brent Truskowski, 9/8/89: Transmittal of Revised Draft Assembly of Alternatives and reply to alternatives analysis letter (discussion of K001 waste and proposed F032 hazardous waste listing).

32. Letter from Richard Fuller to Brent Truskowski, 9/20/89: confirming that EPA is satisfied with use of Tom Aley as a karst expert.

33. Letter from Garrett Bondy to Richard Fuller, 10/10/89: EPA approval of Final Phase II RI/FS Work Plan dated 9/29/89.

34. Letter from Doice Hughes to Richard Fuller (cc: Brent Truskowski), 11/22/89: ADPC&E Permission to conduct Tasks

008758

1 & 2 of tracing study with request for more information before STA granted.

Documents Dealing Specifically with Project Schedule:

1. Letter from Larry Wright to Stewart Braznell, 4/13/88: Letter describing process to determine due dates for submittal of RI/FS documents.

2. Letter from Bob Barker to Larry Wright, 4/19/88: Letter in response to Larry's letter discussing document due dates.

3. Fax from Ruth Izraeli to Richard Fuller and Bob Barker, 6/22/88: Arkwood RI/FS project schedule.

4. Letter from Richard Fuller to Ruth Izraeli, 6/29/88: Proposed changes in intermediate deadlines.

5. Memo from Stephen Doss to Ruth Izraeli (cc: Richard Fuller), 7/26/88: Final revised RI/FS schedule.

6. Memo from Stephen Doss to Ruth Izraeli (cc: Richard Fuller), 9/13/88: Revised RI/FS critical path schedule.

7. Letter from Richard Fuller to Brent Truskowski, 2/2/89: Revised critical path schedule.

8. Fax from Weston, 4/24/89: Revised critical path schedule.

9. Letter from Bob Barker to Brent Truskowski, 5/30/89: Confirmation of agreements reached with regard to delivery of Phase I RI report and Phase II work plan.

10. Letter from Richard Fuller to Brent Truskowski, 6/15/89: Requested changes to the revised critical path schedule received from Weston.

11. Letter from Allyn Davis to Bob Barker, 8/23/89: Proposed second amendment to the Administrative Order on Consent.

12. Letter from Allyn Davis to Bob Barker, 9/19/89: Discusses schedule delays, stipulated penalties, etc.

13. Letter from Allan Gates to Cheryl Mack, 9/20/89: Follow up on his discussion with her regarding the proposed amendment to the consent administrative order.

008759

14. Letter form Allan Gates to Cheryl Mack, 10/4/89: Letter expressing concern over EPA's lack of responsiveness to attempts at resolving scheduling difficulties, etc.

15. Letter from Allan Gates to Allyn Davis and Cheryl Mack, 10/26/89: Chronology of events concerning schedule conflict with MMI's proposal for resolution of matter.

Draft or Final Reports and Plans:

1. Missing monthly progress reports:

   July 1986
   Sept.-Dec. 1986
   Jan. 1987
   Mar. 1987
   June-Aug. 1987
   Oct. 1987
   Dec. 1987
   Feb.-Dec. 1988
   Jan.-Dec. 1989
   Jan.-July 1990

2. From Richard Fuller to Ruth Izraeli, 5/8/87: Updated version of Arkwood Health and Safety Plan.

3. From ERM-Southwest to Region VI EPA, 3/31/89: Six volumes of analytical data plus data index.

4. From Richard Fuller to Brent Truskowski, 4/4/89: Endangerment Assessment (1st Draft).

5. Draft Phase I Remedial Investigation Activities Summary Report to Brent Truskowski, 4/13/89.

6. From Richard Fuller to Brent Truskowski, 5/26/89: Endangerment Assessment (2nd Draft).

7. From Richard Fuller to Brent Truskowski, 6/15/89: Responses to EPA technical comments on 1st draft of Phase I Remedial Investigation Activities Summary Report.

8. From Richard Fuller to Brent Truskowski, 6/15/89: Phase I Remedial Investigation Activities Summary Report, 2nd Draft (Red-line version).

9. Treatability Study Progress Report to Brent Truskowski, 6/29/89.

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10. Draft Phase II Remedial Investigation Work Plant to Brent Truskowski, 7/17/89.

11. From Richard Fuller to Brent Truskowski, 1/8/90: Remedial Investigation Report, 1st draft (red-line version), Vols. I and II.

12. From Richard Fuller to Brent Truskowski, 1/8/90: Feasibility Study Report (1st Draft).

13. From Richard Fuller to Brent Truskowski, 1/8/90: Feasibility Study - Vol. II, Treatability Study Report (1st Draft.)

14. From Richard Fuller to Brent Truskowski, 2/26/90: Remedial Investigation Report, 2nd Draft (Red-line version), Vol. I only.

15. From Richard Fuller to Brent Truskowski, 3/2/90: Feasibility Study Report, 2nd Draft (Red-line version).

16. From Richard Fuller to Brent Truskowski, 3/30/90: Remedial Investigation Report, Final, Vol. II only. (EPA acknowledges Vol. I is in Admin. Record.)

17. From Richard Fuller to Brent Truskowski, 3/3/90: Feasibility Study Report, Intended to be final, Vol. I only. (EPA acknowledges Vol. II in admin. record but they still did not approve Vol. I.)

18. Fax from Steve Calhoun to Brent Truskowski, 4/27/90: Included corrected FS pages and summary of monitor well water producing characteristics.

B. EPA's Proposed Plan of Action

This section respectfully presents a discussion of several factual or technical errors within the EPA Region 6 "Proposed Plan of Action" (PPA) for the Arkwood, Inc. site (July 1990).

Page 2: The Administrative Order on Consent was entered into in May 1986, not 1985.

Page 4: The PPA states that the majority of site risks result from the presence of dioxin. This is incorrect, since the Arkwood Endangerment Assessment has demonstrated that the majority of cancer risk at the main site is due to the carcinogenic polynuclear aromatic hydrocarbons (PNA-C). Even when the dioxin concentrations are calculated according to the revised Toxicity Equivalent Factors, the geometric mean dioxin concentrations do not signifi-

cantly change from those in the original endangerment assessment. If concentrations do not change, exposure and risk remain the same. The original assessment showed that 36 per cent of cancer risk at the main site was due to dioxin, with the remainder (64 per cent) resulting from the PNA-Cs. The EPA bases its statement concerning relative dioxin risk on its own risk assessment for the site. As shown in Section IV - C of this document, however, this assessment is inaccurate and inappropriate to the Arkwood, Inc. site.

Page 9: The PPA states that Alternative H is the only soil remediation option that provides long-term protection to ground water. However, alternatives C, C1, D, E, F, and G provide varying degrees of sludge destruction and soil remediation to a clean-up level protective of ground water. These alternatives were not fully evaluated on this issue in the PPA, but are summarily dismissed on the basis of an improbable catastrophic sinkhole formation.

In addition, within this same paragraph, the PPA states that Alternative H protects the public and the environment to the "maximum extent possible," implying that this criterion renders Alternative H preferable to all others. It should be pointed out that "maximum protection" to the public is not the goal of site remediation. Protection to a reasonable and acceptable level at a cost-effective expenditure of monetary resources is the goal of site remediation. That goal is met by numerous other alternatives put forth in the FS Report.

Page 12: In the definition of "Dioxin," the PPA states that dioxin is a "probable cancer causing [sic] agent." This unsubstantiated comment should be heavily qualified. There is no evidence whatever for the human carcinogenicity of dioxin, despite numerous well-documented cases of human exposure (see Chemrisk documents in Appendices). The only carcinogenic data for dioxin comes from laboratory data on experimental animals.

C. Public Meetings

In accordance with Section 117 of the Comprehensive Environmental Response Compensation and Liability Act, the EPA has periodically held public meetings at the Omaha Public School to disseminate information concerning progress at the Arkwood, Inc. Superfund site. In order to correct and clarify some errors and misconceptions arising from the EPA's comments at the last two public meetings, the following responses are respectfully provided. Each response is preceded by the relevant portion of the EPA's comment as it appears in the transcript of the proceedings. A transcript of the February 12, 1990 public meeting is attached as Appendix G, for incorporation in the Administrative Record.

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February 12, 1990 Public Meeting

Comment (Page 9, Lines 15-18):

Mr. Truskowski: The deepest one we have is a well that was actually already there; it is down a little over a hundred feet. And then we put some deeper ones in the railroad ditch area.

Response:

The railroad ditch monitor wells are all relatively shallow, much shallower than 100 feet. They range in depth from 15 to 23 feet.

Comment (Page 10, Lines 10-12):

Mr. Bondy: Two parts per million.

Mr. Truskowski: What is that, about 0.02 percent, is that how it translates across?

Response:

In terms of percent, the PCP concentration in New Cricket Spring is approximately 0.0002% not 0.02%.

Comment (Page 10, Lines 15-19):

Mr. Truskowski: Right, right, it comes out it comes out of Cricket Springs along the channel, and this water disappears and comes back, and disappears and comes back, and eventually runs into Cricket Creek.

Response:

The water flowing in Cricket Spring Channel has not been observed to repeatedly "disappear and come back" and eventually run into Cricket Creek. Cricket Spring Channel is typically dry all the way from the point at which New Cricket Spring water sinks below the stream bed to its confluence with Cricket Creek.

Comment (Page 15, Lines 1-):

Beige Jacket: How many wells did you test in this area?

Mr. Truskowski: I think we tested somewhere around ten.

Response:

A total of 15 domestic wells were sampled at various times.

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Comment (Page 21, Lines 13-19):

The way we found what wells we found was going through the county, to the city, and a lot of the wells may or may not be registered with the county or the city. So, that's the ones that we have. So, I would say 40, maybe 50, which would give us 25, 20, 25 percent of the wells.

Response:

The initial domestic well survey within the 1.5-mile radius study area identified 36 wells. We sampled 15 of these or 42 percent of the identified wells.

Comment (Page 30, Lines 7-12):

Mr. Bondy: No, we are talking about dioxin.

Mr. Truskowski: Oh, about fourteen parts per million.

Mr. Bondy: Fourteen part per million is the highest we saw.

Response:

They meant to say fourteen parts per billion, not fourteen parts per million.

Comment (Page 44, Lines 11-12):

Mr. Truskowski: Eighty feet in this area is actually a fairly deep well.

Response:

Average domestic well depths in this area are hundreds of feet. Eighty feet is a shallow well for this area.

July 25, 1990 Public Meeting

Comment (Page 13, Lines 6-14):

... treatability study. In the treatability study we looked at the soil washing, which is just what it sounds like, you run the soil through the, for lack of a better word, water in a washing machine. It remediates the pentachlorophenol, however, it fails to remediate the carcinogenic polynuclear aromatic hydrocarbons, or the dioxins.

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Response:

Soil washing is basically a volume reduction process whereby the fine soil particles are separated from the coarse particles. It has been demonstrated that the pentachlorophenol (PCP), polynuclear aromatic hydrocarbons (PAHs) and dioxins tend to concentrate in the fine size fraction. Therefore, by removing the fine size fraction, soil washing remediates all three, not just the PCP.

Comment (Page 13, Lines 15-20):

We also looked at stabilization, in other words, turning the soil into a concrete-like mass, but it failed to remediate the pentachlorophenol or the carcinogenic PNAs, and we don't have the results on the dioxins.

Response:

Stabilization of the affected soil can be achieved in several ways. During the Treatability Study, soil stabilization testing conducted on untreated soils indicated that PCP mobility might be increased by increasing the pH during the stabilization process. However, no such increase in mobility was noted for PNAs. Therefore, stabilization would be effective for carcinogenic PNAs. Although no data was developed for dioxins, available literature, including the proposed Plan of Action for the Texarkana site, indicates that stabilization would also be effective for these compounds.

Comment (Page 13, Lines 21-25):

We also looked at an oxidation, which is a pretreatment step to a bioremedial, biological treatment. However, it failed to show any enhanced results, so it was scrapped early as an...

Response:

Oxidation as a pretreatment step did not provide enhanced biological degradation. However, chemical oxidation did reduce both PCP and PNA levels from 5 to 20 percent in the Arkwood, Inc. site soils. It therefore does appear to have some benefit, but not as a stand-alone treatment.

Comment (Page 14, Lines 1-10):

... idea. And then the biological treatment itself, the biological treatment, basically using the microorganisms in the soil and other microorganisms, it failed to remediate the pentachlorophenol, I'm

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sorry, it did remediate the pentachlorophenol, however, it failed to remediate the carcinogenic PNAs or the dioxins to levels which EPA considered protected.

Response:

Biological treatment studies indicated that it was an effective method of reducing the concentrations of both PCP and PNAs in the sand/fines fraction of the site soil. After 56 days of treatment PCP and PNA concentrations had been reduced by approximately 85 and 80 percent, respectively. PCP concentrations had reached the 300 mg/kg action level being proposed by the EPA. Therefore, biological treatment is an effective remedy for treating both PCP and PNAs. Insufficient data are available to make a similar statement regarding dioxins.

Comment (Page 38, Lines 3-20):

Since February we have done a more detailed review of the treatability study results and a more detailed review of the geology and found one treatability test showed that the biological treatment of the sieve and wash would not remove the contaminants concerned at the levels we felt comfortable with leaving them at the site.

Response:

As stated previously in response to the EPA comment on page 14, lines 1-10, the biological treatment of the sand/fines fraction did result in significant reduction of PCP and PNA concentrations. After 56 days, the PCP concentrations were reduced to the 300 mg/kg clean up level contained in the Proposed Plan of Action. Therefore, the above comment is basically incorrect.

Comment (Page 38, Lines 21-25 and Page 39, Lines 1-3):

Second, we felt that the uncertainty with the geology, the possibility of sinkholes, for example, was such that we could not afford to leave thousands of parts per million of pentachlorophenol and significant quantities of dioxin sitting at the site.

Response:

See Section I of this document.

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APPENDIX A

008767

McLaren Hart

September 13, 1990

Ms. Jean Mescher
Project Manager
McKesson Corporation
One Post Street
San Francisco, CA 94104

Dear Jean:

Enclosed are the following deliverable documents:

- Review of the 1989 Risk Assessment and Development of Refined Health Risk Estimates
- Position Paper Addressing the Scientific Basis of the new TEF for OCDD
- Position Paper Addressing the Inappropriateness of a 20 ppb Cleanup Level for Dioxin at Arkwood
- A Discussion of Precedence at Other SuperFund Sites Regarding Alternatives to Incineration for Soils Containing >20 ppb 2,3,7,8-TCDD Equivalence
- Preliminary Health-based Cleanup Levels for carcinogenic PAHs at Arkwood

16755 Von Karman Avenue, Irvine, CA 92714 (714) 756-2667 FAX (714) 756-8460

008768

The primary contributors to these documents were Brent Finley, Teri Copeland and myself. Please call if you have any questions pertaining to these documents. We look forward to working with you in the future.

Sincerely,

Brent Finley, Ph.D.
Supervising Toxicologist

Teri Copeland, M.S.
Environmental Toxicologist

Mark Harris, Ph.D.
Environmental Toxicologist

ChemRisk Division
McLaren/Hart

Enclosures

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APPENDIX A

Evaluation of the 1989 Endangerment Assessment for Arkwood, Inc. Site

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EVALUATION OF THE 1989 ENDANGERMENT ASSESSMENT FOR ARKWOOD

SEPTEMBER 12, 1990

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TABLE OF CONTENTS

EXECUTIVE SUMMARY ES-1

1.0 INTRODUCTION 1-1

2.0 PROJECT BACKGROUND 2-1

2.1 Site Description and History 2-1
2.2 Previous Investigations 2-1

3.0 SELECTION OF INDICATOR CHEMICALS 3-1

4.0 HAZARD IDENTIFICATION 4-1

4.1 Mechanism of Action of PCDDs/PCDFs and Use of Toxic Equivalent Factors (TEFs) 4-1
4.2 Adverse Health Effects 4-6

4.2.1 Acute Toxicity: Animals 4-6

4.3 Non-Carcinogenic Chronic Toxicity/Biochemical Effects: Animals 4-7
4.4 Acute/Chronic Toxicity: Humans 4-8
4.5 Carcinogenic Effects: Animals 4-9
4.6 Carcinogenic Effects: Humans 4-10
4.7 Metabolism and Disposition of PCDDs and PCDFs 4-11

4.7.1 Metabolism 4-11
4.7.2 Disposition 4-12

4.8 Hazard Identification: Summary 4-13

5.0 DOSE RESPONSE ASSESSMENT 5-1

5.1 Acceptable Daily Intakes 5-2
5.2 Extrapolating Carcinogenic Risks from High-Dosage Bioassays 5-3
5.3 Revised Cancer Potency Factor for TCDD 5-4

6.0 EXPOSURE ASSESSMENT 6-1

6.1 Fate and Transport 6-1
6.2 Pathways and Routes of Exposures 6-3

6.2.1 Exposure Via Ingestion 6-4
6.2.2 Exposure via Dermal Absorption 6-9

6.3 Residence Lifetime 6-12

008772

TABLE OF CONTENTS
(Continued)

7.0 CALCULATION OF EXPOSURE POINT CONCENTRATIONS 7-1

7.1 Test for Log Normality of PCDD/PCDF Data 7-1
7.2 Sample Grids 7-3
7.3 Time-Dependent Contaminant Concentrations 7-3

8.0 DOSE CALCULATION AND HEALTH RISK ESTIMATES 8-1

8.1 Exposure Scenarios 8-1
8.2 General Intake Equation 8-1
8.3 Specific Intake Equations 8-3
8.4 Calculation of Total LADDs and Cancer Risks 8-4

9.0 RISK CHARACTERIZATION 9-1

9.1 Characterization of Non-Cancer Hazards 9-1
9.2 Characterization of Cancer Risks 9-1
9.3 Background Levels of Dioxin Uptake and Body Burden . . 9-5

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EXECUTIVE SUMMARY

This report presents an independent review of the dioxin-related cancer risk estimates presented in the 1989 Endangerment Assessment for the Arkwood site in Omaha, Arkansas. The health risks estimates presented in this review are 2-3 orders of magnitude lower than those estimated in the 1989 EA, primarily due to the use of refined exposure assumptions which have been published in the peer-reviewed scientific literature. The conclusions of this review are as follows:

- Dioxin-related cancer risks for railroad personnel, children, and adults exposed to soils at the main site or in the railroad ditch are in the order of 10^{-6} or lower.
- The health risk estimates presented in the 1989 EA (7×10^{-6} - 2×10^{-5}) should be considered upper-bound estimates of potential cancer risk.
- Estimated rates of dioxin uptake at Arkwood are approximately the same as background levels throughout the U.S.
- Estimated rates of dioxin uptake at the Arkwood site are several orders of magnitude lower than the "acceptable" levels that have been established by several regulatory agencies throughout the world.
- The incremental dioxin body burdens associated with exposure to Arkwood soils would be indistinguishable from pre-existing background levels.

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1.0 INTRODUCTION

This report presents an independent review of the dioxin-related cancer risk estimates presented in the "Endangerment Assessment, Arkwood, Inc., Omaha, Arkansas" (ERM-Southwest, Inc., August 30, 1989). The Arkwood site is a former wood-preservative facility near Omaha, Arkansas. It was in operation from 1962 to 1984; the chemicals used during operation were pentachlorophenol and creosote. The purpose of the 1989 Endangerment Assessment (EA) was to assess the potential health risks associated with exposure to pentachlorophenol, polyaromatic hydrocarbons, and dioxins in soils, groundwater, and surface water at the Arkwood site. The 1989 EA determined that, under the most probable future land use conditions, dioxin related cancer risks could range from 7.5×10^{-6} to 2×10^{-5}. The 1989 EA stated that those risks were within the range of acceptable risks for Superfund sites (10^{-4} to 10^{-7}).

Since the completion of the 1989 EA, several risk assessment guidance documents for Superfund sites have been published by the USEPA, including "Risk Assessment Guidance for Superfund; V.1, Human Health Evaluation Manual" (1989) and "Establishing Target Cleanup Concentrations for Soils at Hazardous Waste Sites" (1989). Several USEPA documents which specifically address appropriate methodologies for assessing dioxin-related cancer risks have also been published including, "Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated Dibenzo-p-Dioxin and Dibenzofurans and 1989 Update" (1989). In addition, several reports have recently appeared in the literature which contain refinements to some of the key assumptions used to assess dioxin-related health risks in the 1989 EA. Some of the refinements include:

- published values for the bioavailability of octachlorinated dioxin,

- new cancer potency factors for dioxin, and
- revised soil ingestion rates for children and adults.

Incorporation of these and other refinements could substantially impact the health risk estimates for the Arkwood site.

The objective of this independent review is to provide refined dioxin risk estimates in a manner consistent with acceptable risk assessment methodologies and the current scientific literature. An independent review such as this one can often be a useful tool for the risk manager when making decisions regarding protection of public health, as it provides an objective professional critique of the validity of the initial risk assessment conclusions. Where appropriate, the refined methodologies used in this review are compared to the risk assessment approach used in the 1989 EA.

The organization of this evaluation follows the guidelines published by the National Academy of Sciences (NAS) in 1983. These guidelines suggest that risk assessments should contain some or all of the following four steps: Hazard identification (the determination of whether a particular chemical is or is not causally linked to particular health affects), dose-response assessment (the determination of the relation between the magnitude of exposure and the probability of occurrence of the health effects in question), exposure assessment (the determination of the extent of human exposures before or after application of regulatory controls), and risk characterization (the description of the nature and often the magnitude of human risk). In addition, other sources will be used for guidance in preparing this evaluation including; the Risk Assessment Guidance for Superfund Volume 1: Health Evaluation Manual (Part A)

published by EPA (Dec. 1989), the EPA Exposure Handbook (July, 1989), and other recent pertinent publications.

The format of this risk assessment evaluation is as follows:

SECTION 2 PROJECT BACKGROUND (location of site, activities at the site etc.)

SECTION 3 SELECTION OF INDICATOR CHEMICALS FOR THIS EVALUATION

SECTION 4 HAZARD IDENTIFICATION (acute, chronic non-carcinogenic and carcinogenic effects, metabolism and disposition, and human body burden)

SECTION 5 DOSE-RESPONSE ASSESSMENT

SECTION 6 EXPOSURE ASSESSMENT (fate of PCDDs and PCDFs, pathways and routes of human exposure, modified intake assumptions)

SECTION 7 CALCULATION OF EXPOSURE POINT CONCENTRATIONS (statistical data analyses)

SECTION 8 DOSE CALCULATION AND HEALTH RISK ESTIMATES (calculation of human intakes and determination of cancer risks)

SECTION 9 RISK CHARACTERIZATION

SECTION 10 REFERENCES

2.0 PROJECT BACKGROUND

The purpose of this section is to briefly review some of the key elements of the Arkwood site history. Most of this information is excerpted from the 1989 EA.

2.1 Site Description and History

The Arkwood site is located 0.5 miles southwest of Omaha, Arkansas in Boone County and is found on the west side of U. S. Highway 65. The site covers approximately 15 acres and is bordered on its northeastern boundary by a branch line of the Missouri Pacific Railroad. Currently, the Arkwood site is not in use, however from 1962 to 1984 the site was used as a wood treatment facility by both Arkwood, Inc. (1962-1973) and Mass Merchandisers Inc. (1973-1984). The primary wood preservatives used at the site during this period (1962-1984) were pentachlorophenol (PCP) and creosote.

2.2 Previous Investigations

ERM-Southwest Inc. was retained in 1987 to conduct the Remedial Investigation and Feasibility Study required by the EPA Region VI 1986 Administrative Order on Consent as amended in 1989. In addition, ERM-Southwest Inc. recently prepared an "Endangerment Assessment" (EA) of the Arkwood site (1989). Previously, several companies had been retained for a variety of services related to the site. McClelland Consulting Engineers, Inc. MCE were the initial consultants and were employed by Mass Merchandisers Inc. (MMI) to advise the company on issues related to the site in 1981.

Recent investigations have shown the presence of polychlorinated dioxins and furans (PCDDs and PCDFs) in soils on the main site and in the nearby railroad ditch. Although there are over two hundred different PCDD and PCDF isomers, much of the dioxin at Arkwood consists of a single isomer: octachlorinated dioxin (OCDD). This isomer is a common contaminant of PCP and is typically found at wood treatment sites. OCDD concentrations detected at the Arkwood site range from 7.2 - 26,000 ppb.

3.0 SELECTION OF INDICATOR CHEMICALS

The focus of this independent review is to evaluate the potential health risks associated with human exposure to the PCDDs and PCDFs distributed in and around the Arkwood site. The PCDD and PCDFs were selected as the chemicals of concern for this review for several reasons, including:

- A recent change in the toxic equivalency factors (TEFs) for a number of the PCDDs and PCDFs present at the site (Barnes, et al., 1989).
- The publication of several papers regarding the behavior of octachlorinated dioxin (OCDD) in biological tissues.

As mentioned previously, OCDD is the most prevalent dioxin isomer in Arkwood soils. The recent increase in the TEF value for OCDD, coupled with the publication of new toxicity, absorption and elimination data pertaining to OCDD, supports a reevaluation of the health risks associated with this specific isomer. Accordingly, for the purposes of this assessment, OCDD will be treated as a separate indicator chemical.

Summary

The 1989 EA treated the entire range of dioxin isomers as a single indicator chemical. In this review, OCDD is treated as a separate chemical of concern due to the fact that OCDD is the predominant isomer in Arkwood soils and 1) the TEF for OCDD has been significantly revised, and 2) studies which specifically address the fate of OCDD in biological tissues have recently been published.

4.0 HAZARD IDENTIFICATION

Hazard identification is defined as the process of determining whether human exposure to an agent could potentially cause an increase in the incidence of an adverse health effect (cancer, birth defects, etc.). It also involves characterizing the nature and strength of the evidence of causation (NAS, 1983).

As described earlier, the main purpose of this review is to evaluate the key assumptions used to assess PCDD/PCDF-related health risks in the 1989 EA. The 1989 EA does not contain a detailed discussion of the adverse health effects associated with PCDD/PCDF exposure. Such a discussion is critical to this reevaluation in light of the additional indicator chemicals (OCDD) addressed in this document. The following sections describe acute, chronic and carcinogenic effects associated with exposure to PCDDs/PCDFs. In addition, a discussion of the mechanism of action as well as the metabolism and disposition of PCDDs/PCDFs is included in this section as they relate directly to the toxicity of PCDD and PCDF congeners, including OCDD.

4.1 Mechanism of Action of PCDDs/PCDFs and Use of Toxic Equivalent Factors (TEFs)

Mechanism of Action

The toxic and biologic responses elicited by PCDDs and PCDFs are proposed to occur via a receptor-mediated mechanism. The receptor protein that mediates the action of PCDDs and PCDFs was identified in 1976 and was termed the aryl hydrocarbon (Ah) receptor (Poland, et al. 1976). To be a true receptor-mediated event, certain criteria must be met, including (Clark, et al., 1988):

- specific binding by prospective ligands
- high affinity binding of the ligand with the receptor
- saturable binding by specific ligands
- correlation with a biological event
- tissue and species specificity

The Ah receptor has been shown to meet all of the above criteria (Goldstein and Safe, 1989; Poland and Knutson, 1982). The Ah receptor is associated with a number of biological responses elicited by PCDDs and PCDFs including; teratogenicity, induction of cytochrome P450-dependent monooxygenases, body weight loss, thymic atrophy, regulation of endocrine systems including estrogen and progesterone receptor levels and immunotoxicity (reviewed in Goldstein and Safe, 1989). In addition, this receptor protein has been detected in a variety of tissues including liver, lung, thymus, duodenum, kidney, spleen, testes, heart, adrenal, human placenta, human squamous cell carcinoma, human breast cancer cells and in some sections of brain (Gasiewicz and Rucci, 1984; Harper, et al., 1988; Harris, et al., 1989a; Manchester, et al., 1987; Okey, et al., 1984; Roberts, et al., 1986).

The mechanism by which PCDDs and PCDFs exert their effects is similar to the mechanism proposed for steroid hormones. PCDDs and PCDFs are lipophilic molecules that passively diffuse across the cellular membrane and into the cytosolic compartment of the target cell (Poland and Knutson, 1982). Once in the cytosol, the congener or ligand binds to the Ah receptor which results in both the transformation of the receptor into a DNA binding protein and the translocation of the ligand-Ah receptor complex into the nucleus of the cell (Harris, et al., 1989a; Harris, et al., 1989b; Okey, et al., 1979; Okey, et al., 1980). The nuclear

ligand-Ah receptor complex then interacts with specific DNA sequences termed dioxin responsive elements (DREs) which results in the initiation of transcription. This mechanism is well characterized for the induction of cytochrome P450-dependent monooxygenases. Specific DREs have been identified in the 5-flanking region of the cytochrome P450 gene and binding of the ligand-Ah receptor complex to DREs has been demonstrated (Israel and Whitlock, 1984; Israel and Whitlock, 1983; Whitlock, 1986; Whitlock, 1987; Whitlock, 1988; Whitlock, 1989; Whitlock and Galeazzi, 1984).

Toxic Equivalent Factors

Several studies have shown that the various dioxin isomers bind to the Ah receptor with different efficiencies. Consequently, the mechanism of action described above dramatically affects the health risk assessment process as it suggests that not all PCDDs/PCDFs are equally potent. This fact complicates the risk assessment process because PCDDs and PCDFs are generally found in the environment as mixtures containing any number of the possible 210 congeners. Because of the difficulties in assessing the human health risk from exposure to mixtures of PCDDs and PCDFs, the USEPA devised a system to assign potency values for the many different congeners present in these mixtures. This system ranks the potency of individual congeners relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) (Barnes, et al., 1989; Safe, et al., 1989a; Safe, et al., 1989b). 2,3,7,8-TCDD was chosen as the standard as numerous studies have identified this congener as the most toxic member of the PCDDs and PCDFs (Poland and Knutson, 1982). These potency values are termed toxic equivalent factors (TEFs) and are derived from in vitro and in vivo data.

The type of data used to derive TEFs includes but is not limited to the following: 1) induction of cytochrome P450-dependent monooxygenases in

both human and rodent cell culture as well as in animal models 2) immunotoxicity in mice 3) body weight loss in a variety of animal models 4) reproductive toxicity in mice and 5) receptor-binding studies. To obtain a 2,3,7,8-TCDD toxic equivalency value for a given congener in a mixture, the TEF value established by EPA is multiplied by the concentration of the individual congener in a mixture. For example, the current TEF for octachlorodibenzo-p-dioxin (OCDD) is 0.001. Therefore, 20,000 ppb of OCDD is theoretically equal to 20 ppb of 2,3,7,8-TCDD (20,000 x 0.001 = 20). This process is then repeated for each individual congener or each separate class of congeners and the values (2,3,7,8-TCDD toxic equivalents or TEQs) summed to arrive at an estimate of the toxic potency of the mixture. In 1989, the EPA revised the 1987 TEFs. A comparison of the values is listed in Table 4-1.

Table 4-1 illustrates the distinct differences between the 1987 and 1989 TEF values prepared by EPA. These differences include; 1) values of zero for all non-2,3,7,8-substituted congeners 2) different TEFs for 1,2,3,7,8- and 2,3,4,7,8-PeCDFs and 3) increasing the TEF value for 2,3,7,8-substituted hexa and hepta congeners. Though these changes represent distinct differences from the 1987 EPA-TEFs, most are unlikely to significantly affect the estimates of human health risks at contaminated sites (with the possible exception of the hepta modification). However, a fourth modification, namely increasing the OCDD/OCDF TEF from zero to 0.001 represents a dramatic departure from the 1987 EPA-TEF values and could significantly impact health risk estimates at sites that contain detectable levels of these compounds.

TABLE 4-1

COMPARISON OF THE 1987 EPA-TEF's AND THE REVISED
1989 I-TEF's FOR PCDD's AND PCDF's

Congener	EPA-TEF's (1987)	I-TEF's (1989)
Mono-,Di-, and TriCDD	0	0
2,3,7,8-TCDD	1.0	1.0
Other TCDDs	0.01	0
2,3,7,8-PeCDDs	0.5	0.5
Other PeCDDs	0.005	0
2,3,7,8-HxCdds	0.04	0.1
Other HxCDDs	0.004	0
2,3,7,9-HpCDDs	0.001	0.01
Other HpCDDs	0.0001	0
OCDD	0	0.001
Mono-,Di-, and TriCDF	0	0
2,3,7,8-TCDF	0.1	0.1
Other TCDFs	0.01	0
1,2,3,7,8-PeCDF	0.1	0.05
2,3,4,7,8-PeCDF	0.1	0.5
Other PeCDFs	0.00	0
2,3,7,8-HxCDFs	0.01	0.1
Other HxCDFs	0.0001	0
2,3,7,8-HpCDFs	0.001	0.01
Other HpCDFs	0.00001	0
OCDF	0	0.001

1 International - TEFs

* Table adapted from Barnes et al., 1989

4.2 Adverse Health Effects

The following sections describe the adverse health effects associated with exposure to 2,3,7,8-TCDD and OCDD. As described in Section 3.0, OCDD is treated as a separate indicator chemical in this reevaluation.

4.2.1 Acute Toxicity: Animals

Acute toxicity is defined as a brief exposure (possibly a single exposure) to high concentrations of a potentially hazardous chemical. Data describing the toxic effects associated with exposure to mixtures of PCDDs and PCDFs is somewhat limited. However, an abundance of data exists describing the toxicity associated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) exposure.

2,3,7,8-TCDD

The lethality associated with 2,3,7,8-TCDD varies widely among different species. Table 4-2 lists some LD_{50} values for exposure to 2,3,7,8-TCDD in various species. In all species, the effects of lethal doses of 2,3,7,8-TCDD generally occur following a 1 week latent period. The species specificity described in Table 4-2 does not reflect differences in the levels of the aryl hydrocarbon (Ah) receptor (Gasiewicz and Rucci, 1984). Other toxic effects that are associated with acute exposure to 2,3,7,8-TCDD include wasting syndrome, thymic involution, immunotoxicity, chloracne and teratogenicity.

Wasting syndrome is one of the most characteristic symptoms of 2,3,7,8-TCDD toxicity following administration of sufficiently high doses (Goldstein and Safe, 1989). While the exact mechanism of toxicity of 2,3,7,8-TCDD is not known, a substantial amount of evidence suggest that

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TABLE 4-2

SELECTED LD_{50}'S FOR 2,3,7,8-TCDD
IN SEVERAL SPECIES*

Species	LD_{50} (µg/kg)
Guinea Pig	0.6 -2
Rat	22 - 45
Monkey	70
Rabbit	115
Mouse	114 - 284
Bullfrog	>1000
Hamster	1154 - 5000

*data taken Goldstein and Safe, 1989

the toxic effects of 2,3,7,8-TCDD and related compounds segregate with the Ah locus (Poland and Glover, 1980; Poland and Knutson, 1982).

OCDD

Acute toxicity data pertaining to OCDD is limited. Schwetz, et al. (1973) reported that administration of as much as 1.0 g/kg of OCDD to rats did not cause significant toxicity.

4.3 Non-Carcinogenic Chronic Toxicity/Biochemical Effects: Animals

Chronic toxic/biochemical effects are associated with long-term exposure to relatively low levels of a potentially hazardous substance.

2,3,7,8-TCDD

Chronic toxic effects have been reported for 2,3,7,8-TCDD. Sprague-Dawley rats fed 0.1 µg/kg/day of 2,3,7,8-TCDD for 2 years exhibited a number of toxic effects including; increased mortality, decreased weight gain, slight depression of erythroid parameters, increased excretion of porphyins and d-aminolevulinic acid, increased serum activities of alkaline phophatase, g-glutamyl transferase and glutamic-pyruvic transaminase. Moreover, histopathologic changes were observed in hepatic, lymphoid, respiratory and vascular tissues (Kociba, et al., 1978).

OCDD

Non-carcinogenic toxic and biochemical effects have also been associated with chronic administration of OCDD to rats. Couture et al (1988) observed treatment-related cytoplasmic fatty vacuolization of the liver in male Fischer 344 rats following chronic administration of OCDD (50 µg/kg; 5 days/week for 13 weeks). Moreover, hepatic cytochrome P450-dependent monooxygenase activity was elevated 40-fold compared to control groups as determined by ethoxyresor of in O-deethylase (EROD) activity (Couture, et al., 1988). Recently, Wermelinger et al. (1990) has also reported that chronic administration of OCDD (800 ppb in feed) resulted in a substantial increase in EROD activity (Wermelinger, et al., 1990).

4.4 Acute/Chronic Toxicity: Humans

2,3,7,8-TCDD

The data describing human exposure to 2,3,7,8-TCDD comes primarily from workers who have been exposed during occupational accidents.

Interpretation of these data should be done with caution as there is uncertainty regarding the exact level of exposure to 2,3,7,8-TCDD. Moreover, workers are generally exposed to a wide variety of contaminants. Toxic responses in humans resulting from occupational exposure to 2,3,7,8-TCDD include; chloracne, porphyria cutanea tarda, hyperpigmentation, altered liver function and neurological problems (Bleiberg, et al., 1964; Moses, et al., 1984; Pazdarova Vejlupkova, et al., 1981; Singer, et al., 1982).

OCDD

There is no data in the literature describing acute/chronic toxicity of OCDD in humans.

4.5 Carcinogenic Effects: Animals

2,3,7,8-TCDD

The results of a two-year chronic oncogenicity feeding study (0.1 µg/kg/day) in rats suggested that 2,3,7,8-TCDD increased the incidence of hepatocellular carcinoma and squamous cell carcinomas of the lung, hard palate/nasal turbinates and tongue. Interestingly, 2,3,7,8-TCDD exposure resulted in a decreased incidence of tumors in the pituitary, uterus, mammary glands, pancreas and adrenal gland. Rats fed lesser doses of 2,3,7,8-TCDD (0.01 and 0.001 µg/kg/day) had reduced responses than those reported for 0.1 µg/kg/day (Kociba, et al., 1978).

Hebert et al. (1990) recently investigated the tumor-promoting ability of 2,3,7.8-TCDD, 2,3,4,7,8-pentachlorodibenzofuran (2,3,4,7,8-PeCDF) and 1,2,3,4,7,8-hexachloro-dibenzofuran (1,2,3,4,7,8-HxCDF) in female HRS/J hairless mice. The results of this study indicated that all 3 congeners

were potent promoters of squamous cell papillomas in animals that were initiated with N-methyl-N'-nitro-N-nitrosoguanidine (MNNG). However, no papillomas were observed in animals not first initi ted with MNNG but treated with the congeners. These data suggest that PCDDs and PCDFs are not complete carcinogens but rather promoters of the carcinogenic process. The order of potency for the 3 congeners was determined to be: 2,3,7,8-TCDD > 2,3,4,7,8-PeCDF > 1,2,3,4,7,8-HxCDD (Hebert, et al., 1990) and is in accordance with established structure-activity relationships (Mason, et al., 1985).

OCDD

The only carcinogenic available pertaining to OCDD were reported by King et al (1973). The effect of the OCDD as a complete carcinogen and as a promoter were investigated using mouse skin tumor formation as endpoint. Swiss-Webster mice (30 female and 30 male) were "painted" 3 times weekly with OCDD (0.2 ml of a 0.2 mg/ml solution for 60 weeks). To test for promotional activity, the mice were first treated with 50 mg of dimenthylbenzathracene (DMBA) prior to exposure to OCDD for 59 weeks. No papillomas or carcinomas were detected in either experimental protocol. However, in the complete carcinogenesis study, 1 subcutaneous tumor was observed in each of the sexes. In the promotional study, 3 subcutaneous tumors were observed in the male mice. This data suggests that OCDD is not an initiator or promoter of carcinogenesis in mice.

4.6 Carcinogenic Effects: Humans

The International Agency for Research on Cancer has classified 2,3,7,8-TCDD as possibly being carcinogenic in humans. However, conflicting data exists in the literature pertaining to the carcinogenic effects of PCDDs and PCDFs in humans. Case-control studies of workers in

Sweden exposed to phenoxy herbicides or chlorophenols reported significantly increased risks of soft-tissue sarcomas and malignant lymphomas (Ericksson, et al., 1981; Hardell, et al., 1981; Hardell and Sandstrom, 1979). However, case-control studies in New Zealand failed to confirm increases in soft-tissue sarcoma in workers exposed to phenoxy herbicides (Smith, et al., 1982; Smith and Pearce, 1986). An NCI case-control study of agricultural use of herbicides in Kansas, demonstrated an association between the use of phenoxyacetic acid herbicides and non-Hodgkins lymphoma (especially with 2,4-D). However, a correlation between these herbicides and soft-tissue sarcoma or Hodgkin's disease was not observed. Interestingly, 2,4-D does not contain 2,3,7,8-TCDD (Hoar, et al., 1986; Keenan, et al., 1989).

OCDD

There are no reports in the literature describing carcinogenic effects of OCDD in humans.

4.7 Metabolism and Disposition of PCDDs and PCDFs

4.7.1 Metabolism

2,3,7,8-TCDD

Several metabolites arising from mammalian metabolism of 2,3,7,8-TCDD have been reported including; 2-hydroxy-3,7,8-trichlorodibenzo-p-dioxin, 2-hydroxy-1,3,7,8-tetrachlorodibenzo-p-dioxin, 1-hydroxyl-2,3,7,8-tetrachlorodibenzo-p-dioxin, diphenyl ethers and 4,5-dichlorocatechol (Neal, et al., 1984; Poiger and Buser, 1984; Poiger, et al., 1982). In vivo and in vitro oxidative metabolism of 2,3,7,8-TCDD does not result in significant adduct formation with RNA, DNA or protein (Guenthner, et al.,

1979; Poland and Glover, 1979). Two metabolites namely, 2-hydroxy-3,7,8-trichloro-dibenzo-p-dioxin and 2-hydroxy-1,3,7,8-tetrachlorodibenzo-p-dioxin have been shown to be non-toxic at doses up to 5000 µg/kg in rats. 2-hydroxy-3,7,8-trichlorodibenzo-p-dioxin induced EROD activity in rats but was at least 3 orders of magnitude less potent than 2,3,7,8-TCDD (Mason and Safe, 1986). These results suggest that metabolism does not play a major role in the toxicity of 2,3,7,8-TCDD.

OCDD

Birnbaum and Couture(1988) recently investigate the metabolism of OCDD in rats. This group reported that extracts from tissues of rats treated with [^{14}C]OCDD revealed a single band of radioactivity which co-migrated with the OCDD standard. In addition, no [^{14}C]CO2 or other radio labeled volatiles were detected following administration of [^{14}C]OCDD (Birnbaum and Couture, 1988). The authors report that some radioactivity was detected in the urine of the rats but due to the low specific activity of the [^{14}C]OCDD confirmation of a metabolite was not possible.

4.7.2 Disposition

2,3,7,8-TCDD

Following a single oral dose of 1.0 µg/kg of [14C]-2,3,7,8-TCDD to rats, ^{14}C activity could be detected in feces but not in urine. Moreover, ^{14}C activity was primarily located in the liver and adipose tissue and the half-life for [^{14}C]-2,3,7,8-TCDD was calculated to be 31 ± 6 days. Repeated oral doses also resulted in the major excretion route being feces though some radioactivity was detected in urine. The half-life following multiple administration of [^{14}C]-2,3,7,8-TCDD was calculated to be 23. 7 days (Rose, et al., 1976). Recently, Abraham et al. (1989) suggested that

the route of exposure dramatically effects the accumulation of PCDDs and PCDFs (in a mixture) in hepatic and adipose tissue. Moreover, the percentage of each congener accumulating in hepatic and adipose tissue varied depending on chlorination. The greater the chlorination the greater the accumulation in hepatic tissue was the general trend for tetra, penta, hexa and hepta substituted congeners. Brewster et al. (1988) recently described the disposition of 2,3,4,7,8-PeCDF in Rhesus Monkey following a single i.v. injection. The congener was cleared rapidly from the blood and was distributed to the liver, skin, adipose and muscle tissues. As noted for 2,3,7,8-TCDD, excretion was primarily via the feces with a minimum whole body half-life of ≈ 38 days (Brewster, et al., 1988).

OCDD

Recently, the disposition of OCDD was investigated in male Fischer 344 rats (Birnbaum and Couture, 1988). As with 2,3,7,8-TCDD and 2,3,4,7,8-PeCDF, the primary excretion route following oral administration was feces. The majority of the administered OCDD that was absorbed was found in liver, adipose and skin tissue. The whole-body half-life following oral administration was predicted to be 3-5 months. Wermelinger et al. (1990) reported that based on liver/adipose concentration ratios, OCDD preferentially accumulates in hepatic tissue of rats. Interestingly, OCDD was found to partition into hepatic tissue to a greater extent than 2,3,7,8-TCDD.

4.8 Hazard Identification: Summary

The 1989 EA used the 1987 TEFs to determine 2,3,7,8-equivalence in soil. This review will use the updated 1989 TEFs. 2,3,7,8-TCDD, the most characterized member of the PCDDs and PCDFs, exhibits potent acute and

chronic toxicity in animal models. Epidemiological data describing human acute/chronic toxic effects caused by 2,3,7,8-TCDD is weak but does suggest some non-cancer health hazards. Treatment of animals with 2,3,7,8-TCDD has been associated with the occurrence of a variety of neoplastic lesions. However, epidemiology studies are inconclusive in determining whether or not 2,3,7,8-TCDD is indeed a human carcinogen. Nevertheless, IARC has labeled 2,3,7,8-TCDD as a probable human carcinogen. Metabolites are thought to play little or no role in the toxicity of 2,3,7,8-TCDD and no metabolites were detected in OCDD treated rats. As predicted by structure-Ah receptor binding relationships (Mason, et al., 1986), OCDD is substantially less potent than 2,3,7,8-TCDD. However, multiple administration of this congener results in several "dioxin" like responses including: 1) the induction of cytochrome P450-dependent monooxygenase activity (EROD activity) and 2) mild hepatotoxicity. Moreover, this congener tends to accumulate in rat hepatic and adipose tissue.

5.0 DOSE RESPONSE ASSESSMENT

Dose-response assessment is the process of characterizing the relationship between the dose of an agent and the anticipated incidence of an adverse health effect in an exposed population (Preuss and Ehrlich, 1987). The bulk of our knowledge about the dose-response relationship is based on data collected from animal studies and theoretical precepts about what might occur in humans. Moreover, mathematical models are used to estimate the possible response at levels below those tested in animals. Limitations of the models include; 1) uncertainty in extrapolation of results obtained from animal data to the human population and 2) extrapolating responses obtained from high-dose studies to estimate responses at very low doses.

Historically, there have been two approaches to estimating risk levels from exposure to toxic agents. One approach involves calculating an acceptable daily intake (ADI) by applying a safety factor to doses of the agent that did not produce an observed effect in laboratory animals (no observed effects level, NOEL). This approach is typically used for noncarcinogenic toxic effects. The second approach relies on the use of mathematical models to extrapolate experimental dose-response information obtained from animal exposure studies using high dosage levels to the low levels likely to encountered by human populations. This latter approach is often used for carcinogens and generally assumes that there is not threshold for the response being extrapolated; that is, any dose, no matter how small, will pose some risk.

The 1989 EA does not contain a detailed discussion regarding dose-response assessment of PCDD and PCDF effects. Such a discussion is critical to this reevaluation in light of the revised dose response criteria used to derive the refined cancer risk estimates in this document. The following

sections discuss dose response assessment of non-cancer and cancer effects of dioxin.

5.1 Acceptable Daily Intakes

As discussed previously, 2,3,7,8-TCDD has a variety of toxic effects on laboratory test animals, including embryotoxicity and tetratogenicity. Effects have also been observed in various organs and systems including the thyroid, liver, and skin, as well as the immune system. Only the 2,3,7,8-TCDD and a mixture of HCDD isomers have been demonstrated to be animal cacinogens.

Normally, an ADI is not derived from a NOEL for compounds that have been found to be carcinogenic. However 2,3,7,8 has not been proved to be an initiator of neoplastic growth. In light of this fact, several agencies have found it appropriate to use a NOEL to calculate a guideline or standard. For 2,3,7,8-TCDD, a NOEL of 1 ng/kg of body weight per day in rats has been reported in both a three-generation reproduction study by Murray et al. and a 2-year oncogenic study by Kociba. On the basis of these studies, regulatory agencies in Canada, The Netherlands, Germany, and elsewhere have developed guidelines or standards by applying safety factors ranging from 100 to 1000 to the NOEL. A NOEL of 0.18 μg/Kg-day has been reported for a mixture of HCDDs tested in rats. NOELs have not been determined for other PCDD and PCDF isomers. A list of the guidelines established by regulatory agencies or published by various authors using the ADI/NOEL approach is provided in Table 5-1.

TABLE 5-1

ADI's FOR 2,3,7,8-TCDD USED
BY VARIOUS REGULATING AGENICIES

Agency	Basis	ADI
Canada	NOEL of 1 ng/kg-day safety factor of 100	10 pg/Kg-day
The Netherlands	NOEL of 1 ng/Kg-day safety factor of 250	4 pg/Kg-day
Kimbrough (CDC)	LOEL of 1.8 ng/kg-day safety factor of 1000	4 pg/Kg-day
NYSDOH	NOEL of 1 ng/Kg-day safety factor of 500	2 pg/Kg-day
West Germany	NOEL of 1 ng/Kg-day Safety factor of 100-1000	1-10 pg/Kg-day

5.2 Extrapolating Carcinogenic Risks from High-Dosage Bioassays

Regulatory agencies have generally assumed that agents which are carcinogenic should be treated as if they do not display thresholds. To estimate the theoretically plausible response at these low doses, various mathematical models are employed. The USEPA generally uses the linearized multistage model for low dose extrapolation (Munro and Krewski, 1981). This model assumes that the effect of the carcinogenic agent on tumor formation as seen at high doses in animal data is basically the same at low doses, i.e., the slope of a dose-response curve can be extrapolated downward in a linear manner.

The USEPA, the Centers for Disease Control (CDC) and the Food and Drug Administration (FDA) have performed dose-response assessments of 2,3,7,8-TCDD. The USEPA and CDC used the linearized multistage model to estimate the dose-response relationship for low-levels of human exposure and is based on data obtained in rodents at high doses. In contrast, the FDA utilizes the linear interpolation model. The purpose of these models is to estimate the maximum possible linear slope (the 95% upper confidence limit) of the dose-response curve in the low dose range. Therefore, it is likely that these models overestimate rather than underestimate the actual cancer risk posed by 2,3,7,8-TCDD and related congeners. The cancer potency figure used by the USEPA is 1.56×10^5 $(mg/kg/day)^{-1}$ and was produced by the multistage model. The data input into the model was based on the geometric mean of two pathologists (Dr. Kociba and Dr. Squire) interpretations of tissue slides from Kociba et al. (1978) data for female rats using pooled tumor types (Keenan, et al., 1989).

It is important to emphasize that use of the linearized multistage model is likely to be inappropriate for low dose extrapolations involving 2,3,7,8-TCDD, including: 1) the model forces linearity at low doses, linearity should not be applied to chemicals that act through a receptor-mediated event, and 2) the model does not account for the reversible behavior of promoters or the "threshold" concept that applies to promoter action (Paustenbach et al. 1986). As noted above, it appears as if 2,3,7,8-TCDD is a promoter rather than an initiator of the carcinogenic process (Hebert et al., 1990).

5.3 Revised Cancer Potency Factor for TCDD

Recently, the tissue slides from the Kociba et al. (1978) study were reevaluated by an independent Pathology Working Group (PWG) using the current NTP classification scheme for proliferative lesions in rodent

hepatic tissue (Keenan, et al., 1990). The updated guidelines now distinguish between hyperplasia and an adenoma. Hyperplasia is generally regarded as a non-neoplastic response to degenerative changes in the liver while an adenoma is a benign condition in which the cells in question are still clearly differentiated. The reevaluation of the Kociba et al. (1978) data produced significant differences in the type of tumors detected as well as the number to tumors detected. Based on this reevaluation, Keenan et al. (1990) has recalculated the cancer potency factor (CPF) for 2,3,7,8-TCDD. Depending on the variables used (total hepatic lesions vs hepatocellular carcinoma and overall incidences of lesions vs survival-adjusted incidences), revised CPF values ranged from 1500 to 9700 $(mg/kg/day)^{-1}$. These values suggest that 2,3,7,8-TCDD is 16-100 times less potent than originally thought. These revised values are gaining acceptance in the regulatory community. Recently, the state of Georgia, with support from the CDC, raised the ambient water quality criteria for dioxin based on the new CPF of 9,700 $(mg/kg/day)^{-1}$. For the purposes of this review a CPF of 9,700 $(mg/kg/day)^{-1}$ will be used to assess dioxin related health risks.

Dose-Response Assessment Summary

Normally, an ADI is not derived from a NOEL for compounds that have been found to be carcinogenic. However, 2,3,7,8-TCDD has not been shown to be an initiator of neoplastic growth. In light of this fact, several agencies have found it appropriate to use a NOEL to calculate a guideline or standard (ranging from 1-10 pg/Kg-day). The USEPA has used dose-response assessment to estimate the carcinogenic potency of 2,3,7,8-TCDD. Original estimates suggest that the cancer potency factor for 2,3,7,8-TCDD was 1.56×10^5 $(mg/kg-day)^{-1}$. A revaluation of the Kociba et al (1978) data indicates that 2,3,7,8-TCDD is a much less potent carcinogen than originally suggested. The cancer potency factor (CPF) is more likely in

the range of 1500-9700 $(mg/kg\text{-}day)^{-1}$. A CPF of 9,700 $(mg/kg/day)^{-1}$ is gaining acceptance within the regulatory community. This value will be used to evaluate PCDD/PCDF health risk at the Arkwood site.

6.0 EXPOSURE ASSESSMENT

Exposure assessment, as defined by the National Academy of Sciences (1983), is the process of measuring or estimating the intensity, frequency, and duration of human exposure to an agent currently present in the environment. "In its most complete form, exposure assessment should describe the magnitude, duration, schedule, and route of exposure; the size, nature, and classes of the populations exposed; and the uncertainties in all estimates" (NAS, 1983). Accordingly, this section of the review discusses the manner in which the PCDDs and PCDFs distribute and degrade in the environment (Section 6.1) and the estimated frequency of contact between potential visitors to the site and the environmental media (Section 6.2). These exposure estimates, when combined with site-specific environmental PCDD and PCDF concentrations, provide the basis for assessing PCDD and PCDF uptake and health risks (Section 6.7). Many of the exposure estimates presented in Section 6.2 are refinements of the assumptions used in the 1989 EA. These refinements are based primarily on literature reports which have been published since completion of the EA. Table 6-1 contains a summary of the exposure parameters used in this assessment and provides a comparison to the values used in the 1989 EA.

6.1 Fate and Transport

PCDDs and PCDFs are lipophilic molecules and as such are very insoluble in water. Moreover, PCDDs and PCDFs are resistant to a variety of harsh conditions including acid, base and heat treatments. It has been estimated that 99.99% destruction of tetra-substituted molecules occurs when temperatures reach 977° C (Shaub and Tsang, 1983). PCDDs and PCDFs have been shown to undergo photolytic degradation. However, the photolytic decomposition of PCDDs and PCDFs is extremely dependent on the surrounding

conditions i.e., solvents used and the presence of hydrogen donors (Dobbs and Grant, 1979; Hung and Ingram, 1990). Interestingly, photolysis of OCDD resulted in the replacement of a lateral Chlorine atom (2,3,7,8-positions) with a Hydrogen atom which suggests that the formation of 2,3,7,8-TCDD from higher chlorinated congeners (i.e., OCDD) does not occur easily under conditions used in the laboratory (Hung and Ingram, 1990). 2,3,7,8-TCDD exhibits a vapor pressure that would suggest reduced volatility (2.02×10^{-7} Pa at 25° C), however this compound does volatilize at ambient soil temperatures (Freeman and Schroy, 1985). 2,3,7,8-TCDD in the vapor phase is predicted to photolyze rapidly with a suggested half-life of 58 min. (Podoll, et al., 1986). 2,3,7,8-TCDD has a soil partition coefficient on the order of 10^6 and thus binds tightly to soil (Burg, 1988). Moreover, the extent of binding of 2,3,7,8-TCDD to soils may be affected by such factors as the percentage of carbon in the soil, the particle size, and the particle morphology (Barnes, 1983).

The 1989 EA assumed that dioxin concentrations in Arkwood soils remain constant over time. Recently, Cerlesi et al. (1989) calculated the half-life of 2,3,7,8-TCDD in soil at Seveso, Italy. Seveso is the site of an explosion which occurred during the synthesis of 2,4,5-trichlorophenol (2,4,5-T) at the Givaudan-Hoffman-LaRoche ICMESA plant. It was calculated that approximately 1.2 kg of 2,3,7,8-TCDD was deposited in and around the plant area. Cerlesi et al. (1989), using data obtained from this site, suggested a half-life of 2,3,7,8-TCDD in soil of 6.2 - 16 years (Cerlesi, et al., 1989). These results are comparable to other half-life determinations of 2,3,7,8-TCDD in soil (Kearny, 1972; Young, 1983). For the purposes of this review, a soil half-life of 16 years will be used to calculate soil concentrations for the various age groups considered in this assessment.

Fate and Transport Summary

The 1989 EA assumed that soil concentrations of PCDDs/PCDFs remained constant over a 70 year exposure. However, 2,3,7,8-TCDD and OCDD have been shown to be susceptible to photolysis and a review of the literature indicates that dioxin in soil degrades with a half-life of 16 years or less. For the purpose of this review, the PCDDs and PCDFs will be assumed to have an environmental half-life of 16 years.

6.2 Pathways and Routes of Exposures

Exposure pathways are the means through which an individual may come into contact with a chemical in the environment (e.g., drinking contaminated water from a well). Routes of exposure describe the means through which the chemical gains entry to the body via a particular pathway (e.g., dermal absorption of a soil-bound chemical while gardening).

This section describes all of the potential pathways and routes of human exposure to the PCDDs and PCDFs at the Arkwood site. The quantitative assessment of exposure, combined with a knowledge of the PCDD and PCDF concentrations present at the site, provides the basis for estimating daily PCDD and PCDF uptake and any associated health risks. Since PCDDs and PCDFs were not detected in the groundwater or surface water at the site, the 1989 EA identified two potential pathways of exposure to site-related PCDDs and PCDFs: dermal and oral absorption from soil. Accordingly, these pathways will be addressed quantitatively included in this review.

6.2.1 Exposure Via Ingestion

Soil Ingestion Rates

Exposure to PCDDs and PCDFs at the Arkwood site may occur through ingestion of contaminated soil either during recreational or occupational activities. Ingestion of soil generally is confined to children between the ages of 1.5 to 6 years of age and this is a result of the tendency of children at this age to mouth and chew foreign objects. Moreover, several factors can influence this behavior including nutritional and economic status as well as race (Charney, et al., 1980). A variety of estimates have been proposed for the amount of soil ingested by young children. Originally, the CDC proposed as much as 10 g/day of soil was ingested by children between the ages of 1 and 3.5 while children 3.5 and 5 years of age were assumed to ingest 1 g/day of soil. However, Paustenbach et al. (1986) calculated that if these assumptions by CDC were correct, then 80% of the entire lifetime dose of a non-volatile, hydrophobic chemical present in soil would occur during the first 5 years of life (Paustenbach, et al., 1986). Other groups have attempted to estimate the soil intake by children including: NRC, 40 mg/day; Day et al. (1975), 100 mg/day; Bryce-Smith (1974), 33 mg/day; Hawley (1985), 100 mg/day.

The 1989 EA assumed that children ingest 200 mg/day and adults ingest 100 mg/day, based on values suggested in the USEPA's 1988 Superfund Exposure Assessment Manual. However, recent reports suggest that soil ingestion occurs at a much lower rate. Recently, Calabrese et al. (1989) conducted a rigorous study to determine the amount of soil that was typically ingested by children. This group utilized the measurement of tracer elements in the feces of 64 healthy children between the ages of 1 and 4 years. This study was more definitive than prior investigations because it analyzed the diet of the children, assayed for the presence of tracers

in the diapers, assayed house dust and surrounding soil, and corrected for the pharmacokinetics of the tracer materials. Eight tracer elements were measured in this study though three elements namely, Si, Al and Y gave the best percentage of recovery (close to 100%) and the lowest standard deviation. Based on the results of two of the three most reliable tracers, the amount of soil ingestion by children (2-4 years of age) was found to average approximately 25 mg/day.

Adults do not generally ingest soil and thus their intake of potentially contaminated soil would be quite low (Vermeer and Frate, 1979). Moreover, even taking into account poor hygiene and eating soil-contaminated food, the 100 mg/day figure suggested by CDC for use in estimating soil intake by adolescents and adults seems high. A figure of 5 mg/day for adolescents and adults is reasonable if, as some have suggested, adults ingestion 10% of the amount of dirt eaten by children. Therefore, the following soil ingestion rates will be used in this review.

Age Group (years)	Soil Ingestion Rate (mg/day)
0-6	25
6-12	5
12-70	5

The ingestion rate of 25 mg/day is derived from Calabrese's (1989) tracer study, and the 5 mg/day value is based on the assumption that adults and adolescents ingest soil at a rate of 10% that of children.

Oral Bioavailability of 2,3,7,8-TCDD

The estimate of oral bioavailability of soil-bound PCDDs and PCDFs is an important parameter in the risk assessment process. Numerous investigators have reported a variety of bioavailability figures for soil-bound 2,3,7,8-TCDD. For example, Lucier et al. (1986) suggested the bioavailability of 2,3,7,8-TCDD was dose dependent: 24% uptake at a dose of 1 µg/kg and 50% at 5 µg/kg (Lucier, et al., 1986). Moreover, Umbreit et al. (1986) reported that bioavailability of 2,3,7,8-TCDD varied between sample sites. For example, a bioavailability of 0.05% for 2,3,7,8-TCDD was reported for soil taken from a New Jersey manufacturing site whereas soil taken from a salvage yard in Newark had an oral bioavailability of 21%. These differences are likely due to the varying levels of organic content of the soils from different sites. As mentioned earlier, the affinity of dioxin for soil increases as the organic content of the soil increases. Interestingly, if the organics were removed from the soil taken from the New Jersey manufacturing site the bioavailability increased to approximately 23%. Recently, Shu et al. (1988) suggested an appropriate oral bioavailability factor for 2,3,7,8-TCDD was 43% (Shu, et al., 1988a). An oral bioavailability factor of 50% was used for all PCDDs and PCDFs in the 1989 EA. In this review, an oral bioavailability factor of 50% will be used for all dioxin isomers except OCDD and OCDF. As described below, animal exposure studies have shown that the oral bioavailability of OCDD and OCDF is much lower than that of 2,3,7,8-TCDD.

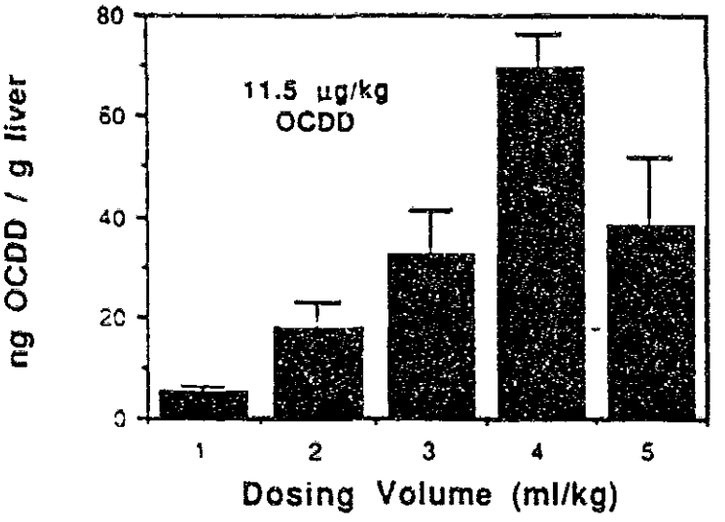

Figure 1. Volume-dependent absorption of OCDD. The vehicle was corn oil and a constant dose of 11.5 µg/kg was used for all volumes tested.

Oral Bioavailability of OCDD

Some recent studies indicate that the oral bioavailability of OCDD is much lower than that of 2,3,7,8-TCDD and other PCDDs and PCDFs (Birnbaum & Couture, 1988). Oral absorption of OCDD suspended in corn oil was reported to be non-linear between doses of 500 and 5000 µg/kg. Moreover, never was more than 10% of the administered dose absorbed by the gastrointestinal tract. A subsequent report (Birnbaum and Couture, 1988) suggested that oral absorption of OCDD was dependent on the volume of the carrier material (in this case corn oil) used. Figure 1 illustrates the absorption of 11.5 µg/kg of OCDD administered in different volumes of corn oil to male Fischer 344 rats.

This data illustrates that as the dosing volume increases (the amount of corn oil OCDD is suspended in) the absorption or bioavailability of OCDD increases. The absorption of OCDD in the 5 ml/kg treatment group is approximately 7-fold greater than the 1 ml/kg treatment group. The same effect was observed using a multiple dosing protocol (data not shown). To summarize the work of Birnbaum & Couture:

- Oral absorption of OCDD suspended in corn oil was found to never exceed 10%.
- OCDD oral absorption increased as the volume corn oil used to suspend OCDD increased. The largest absorption was observed at a volume of 5 ml/kg.

Give the relatively small volumes of soil ingested on a daily basis, it would seem appropriate to use a bioavailability value of much less than 10% for OCDD. The USEPA, in "Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated Dibenzo-p-dioxins and -Dibenzo furans" acknowledges the limited bioavailability of the highly

chlorinated dioxin isomers and suggests that this be considered when assessing environmental health risks:

> "The increased TEF for OCDD/F ignores the issue of relative bioavailability of CDD/CDF congeners, which have not been thoroughly investigated. Lower relative bioavailability of the hepta- and octa- forms compared to the tetra- forms would generally reduce the concern for TEQ estimates for samples such as those which are dominated by the hepta- and octa- forms. Research in this area is needed to resolve this point.'
>
> 'In samples taken from biological organisms exposed to PCP-contaminated soils in Region IX, the TEQs were within a factor of two of each other, when calculated by the I-TEF/89 method or the EPA-TEF/87 method. Although the data are limited, they appear to suggest that the differences in TEQs observed in the PCP-contaminated soil samples are not observed in tissues or organisms exposed to this soil."

These above observations are critical to an accurate assessment of health risks at sites that are principally contaminated by the highly chlorinated dioxin isomers.

Based on the results of Birnbaum and Couture's studies, an oral bioavailability factor of 1% will be used for ingestion of soil bound OCDD. The use of this factor takes into account the following: 1) the data described above that suggests that the bioavailability of pure OCDD is less than 10%, and 2) soil-bound OCDD is likely to be much less bioavailable than OCDD suspended in corn oil.

6.2.2 Exposure via Dermal Absorption

Bioavailability

A number of parameters influence the degree of bioavailability of PCDDs and PCDFs including: aging of the soil, soil type, contaminants and the

PCDD/PCDF concentration in the soil. There are several reports in the literature describing the dermal absorption of 2,3,7,8-TCDD in rats. Poiger and Schlatter (1980) observed that as the dermal dose of 2,3,7,8-TCDD (in soil) increased, the liver concentrations of 2,3,7,8-TCDD increased from 0.05 to 2.2% of the administered dose (Poiger and Schlatter, 1980). Kimbrough et al. (1984), based on the Poiger and Schlatter (1980) study estimated a dermal bioavailability of 1% for humans. Shu et al. (1988) also investigated the dermal bioavailability of 2,3,7,8-TCDD in the rat. This group concluded that dermal absorption of 2,3,7,8-TCDD was time dependent as dermal penetration following 4 hours of skin contact was 60% of that following 24 hour of contact ($p < 0.05$). Moreover, this group indicated that dermal absorption of 2,3,7,8-TCDD following 24 hours of skin contact was approximately 1%. However, the authors state that this estimate is very likely to overestimate human exposure to 2,3,7,8-TCDD via skin, since there is general agreement among researchers that rat and rabbit skin are more permeable to many chemicals than human skin (Shu, et al., 1988b).

Specifically, several reports have suggested that rat skin may be at least 10-fold more permeable than human skin (Bartek, et al., 1972; Bartek and La Budde, 1975). Moreover, it has been reported that the difference in permeability between rat and human skin was greater for lipid-soluble compounds than for water-soluble compounds. Comparison of the permeability of several compounds including the lipophilic haloprogin and water-soluble acetylcysteine gave interesting results. The rat:human dermal penetration ratio for the lipophilic haloprogin was 9:1. In contrast, the very water soluble acetylcysteine rat:human dermal penetration ratio was 1.4:1 (Bartek, et al., 1972; Bartek and La Budde, 1975). These results suggest that absorption by human skin of lipophilic chemicals is approximately 10-fold less than that of rat skin.

The 1989 EA assumed a dermal bioavailability of 1% for PCDDs/PCDFs in soil, based on the Kimbrough estimate. A dermal bioavailability factor of 0.1% will be used in this review for all PCDDs and PCDFs, including OCDD and OCDF. As described above, a 1% dermal absorption of soil-bound 2,3,7,8-TCDD in rat skin is likely to be equivalent to an absorption of 0.10% for humans. A value of 0.10% dermal absorption for OCDD is consistent with the observation that dermal absorption of non-volatile chemicals is typically at least an order of magnitude less than the degree of oral absorption.

Soil Adherance

Another parameter which influences the dermal absorption of PCDD and PCDF, is the amount of soil adhering to human skin. The 1989 EA used a soil adherance factor of 2.0 mg soil per cm^2 of skin, based on values suggested in the USEPA's 1988 Superfund Exposure Assessment Manual. However, several other studies suggest that soil adheres to skin to a much lesser degree. Lepow et al. (1975) using adhesive tape to sample a defined area of skin suggested that the amount of soil adhering to human skin was approximately 0.5 mg/cm^2 (Lepow et al., 1975). Furthermore, the California Department of Health Services Toxic Substances Control Division estimated that approximately 0.9 mg/cm^2 of soil adheres to the hands of children (average age was 11) (Services, 1986). In addition, several studies have been published which describe the effect of soil particle size and organic content of soil on adherence of soil to human skin. For example, Que Hee et al. (1985) using a variety of soils of different particle size suggested that on average, about 0.2 mg/cm^2 of soil was adhering to the hands of small adults (Que Hee, et al., 1985). Driver et al. (1989) surveyed various soils of different organic content for their ability to adhere to human hands (adult male). It was determined that the average amount of soil adhering to human hands was 0.6 mg/cm^2 (unsieved).

Thus, the available literature suggests that the amount of soil adhering to the hands of humans is between 0.2 and 0.9 mg/cm^2 (Driver, et al., 1989). Therefore, for this review, a value of 0.5 mg/cm^2 seems a reasonable estimate for the amount of soil adhering to the human skin.

6.3 Residence Lifetime

The 1989 EA assumed that the same individual could visit the site on a regular basis throughout a 70-year lifetime. Presumably, such an individual would be a nearby resident. As described in the 1988 Superfund Exposure Assessment Manual, 95% of the general U.S. population remains in a single residence for 30 years or less. Consequently, for the purposes of this review, it will be assumed that the duration of regular visits to the site could be no more than 30 years.

Table 6-1 contains a summary of the exposure parameters used in this assessment and in the 1989 EA.

TABLE 6-1

COMPARISON OF EXPOSURE ASSUMPTIONS USED IN 1989 ENDANGERMENT ASSESSMENT AND THE 1990 ENDANGERMENT ASSESSMENT REVIEW

Exposure Factor	1989 EA	1990 Review	Reference Cited for Refined Assumption
Soil Ingestion Rate			
6-12 years	200 mg/day	5 mg/day	Calabrese et al., 1989
12-70 years	100 mg/day	5 mg/day	
Soil Adherence Factor	2 mg/cm^2	0.5 mg/cm^2	Driver et al., 1989
Exposure Duration	70 years	30 years	USEPA, 1989b
Skin Surface Area			
12-70 years	910 cm^2	840 cm^2	USEPA, 1989b
Soil Half-Life, TCDD	---	16 years	Cerlesi et al., 1989 Kimbrough et al., 1984
Cancer Potency Factor	1.56 x 10^5 (mg/kg/day)	9700 (mg/kg/day)$^{-1}$	Keenan, 1990
TEFs	1987 TEFs	1989 TEFs	
Dermal Bioavailability	1%	0.1%	Shu et al., 1988 Bartek et al., 1972 Bartek and La Budde, 1975
Oral Bioavailability	50%	OCDD (1%) Non-OCDD (50%)	Birnbaum and Couture (1988)

[1] All other exposure assumptions are identical to those used in the 1989 Endangerment Assessment.

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7.0 CALCULATION OF EXPOSURE POINT CONCENTRATIONS

This section describes the methodologies used to derive representative soil concentrations.

7.1 Test for Log Normality of PCDD/PCDF Data

Soil samples for PCDD/PCDF analyses were taken from three areas, or sample grids, which were identified as B, TC and RC. The entire main site is identified as the B grid. The TC grid, a former treatment area, is a small area which lies within the main site, and represents approximately 1.3 percent of the main site. RC samples were taken from the small railroad ditch area, located just outside the northern boundary of the site. Table 7-1 contains a summary of the isomer concentrations in the surficial samples taken from each grid. 2,3,7,8-equivalences for OCDD/OCDF and non-OCDD/OCF isomers are listed, using the 1989 TEFs.

As described in the 1989 EA, environmental data are usually lognormally distributed. This is often due to the presence of a significant number of non-detect values in the data set. When data are lognormally distributed, the geometric mean is the appropriate measure of the central tendency of the data. When data are normally distributed, the arithmetic mean is the appropriate measure of the central tendency of the data.

In the 1989 EA, PCDD/PCDF concentrations in surface soils were not tested for lognormality, but were assumed to be lognormally distributed and geometric mean concentrations were used to assess dioxin-related health risks. However, in general, a fairly robust data set (at least 10 samples) is required to conclusively demonstrate lognormality. The limited number of surficial samples from the TC grid (3) and the RC grid

TABLE 7.1
CALCULATION OF 2,3,7,8-TCDD TOXIC EQUIVALENTS

Congeners	1989 TEFs	Sample B-7		Sample B-11		Sample B-17		Sample B-25	
		concen. (ppb)	2,3,7,8 TCDD (TEQs)	concen (ppb)	2,3,7,8-TCDD (TEQs)	concen (ppb)	2,3,7,8 TCDD (TEQs)	concen. (ppb)	2,3,7,8 TCDD (TEQs)
		BC Grid (0 - 6")		BC Grid (0 - 6")		BC Grid (0 - 6")		BC Grid (0 - 6")	
non 2,3,7,8-TCDFs	0	nd	-	0.047	0	nd	-	nd	-
2,3,7,8-TCDF	0.1	nd	-	nd	-	nd		nd	-
non 2,3,7,8-PeCDFs	0	3.7	0	3 88	0	nd	-	8.8	0
1,2,3,7,8-PeCDF	0.05	nd	-	0.42	0.021	nd	-	nd	-
2,3,4,7,8 PeCDF	0.5	nd	-	0.3	0.15	nd	-	nd	-
non 2,3,7,8-HxCDFs	0	128.7	0	42.36	0	70	0	257.7	0
1,2,3,4,7,8-HxCDF	0.1	6.7	0 67	3.2	0.32	nd	-	15	1.5
1,2,3,6,7,8-HxCDF	0.1	nd	-	0 88	0.088	nd	-	nd	-
2,3,4,6,7,8-HxCDF	0.1	nd	-	0 36	0.036	nd	-	nd	-
1,2,3,7,8,9-HxCDF	0.1	4.6	0.46	1.2	0.12	nd	-	7.3	0.73
non 2,3,7,8-HpCDFs	0	633	0	166.9	0	408	0	1064	0
1,2,3,4,6,7,8-HpCDF	0.01	130	1.3	38	0.38	80	0.80	210	2.1
1,2,34,7,8,9-HpCDF	0.01	17	0 17	5 1	0.051	12	0.12	26	0.26
OCDF	0.001	1200	1.2	390	0.390	700	0.7	1600	1.6
TEQs PCDFs			9.80		1.556		1.62		6.19
non 2,3,7,8-TCDDs	0	nd	-	nd	-	nd	-	nd	
2,3,7,8-TCDD	1	nd	-	nd	-	nd	-	nd	
non 2,3,7,8-PeCDDs	0	nd	-	0 03	0	nd	-	nd	
1,2,3,7,8-PeCDD	0.5	nd	-	0 22	0.11	nd	-	nd	
non 2,3,7,8-HxCDDs	0	38	0	8 52	0	26.4	0	51.3	0
1,2,3,4,7,8-HxCDD	0.1	6	0.6	0.98	0.098	nd	-	7.7	0.77
1,2,3,6,7,8-HxCDD	0 1	26	2.6	6.5	0.65	16	1.6	37	3.7
1,2,3,7,8,9-HxCDD	0.1	10	1	2	0.2	3 6	0.36	14	1.4
non 2,3,7,8-HpCDDs	0	500	0	140	0	370	0	700	0
1,2,3,4,6,7,8-HpCDD	0.01	1200	12	330	3.3	730	7.3	1400	14
OCDD	0.001	7800	7.8	2000	2	4300	4.3	8100	8 1
Total TEQs OCDD/OCDF			9.0		2.39		5.00		9.70
TEQs NON-OCDD/OCDF			18 8		5 52		10.18		24.46
TOTAL 2,3,7,8-TCDD TEQS			**27.8**		**7.91**		**15.18**		**34.16**

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TABLE 7.1 (cont)

CALCULATION OF 2,3,7,8-TCDD TOXIC EQUIVALENTS

Congeners	1989 TEFs	Sample B-28 concen. (ppb)	Sample B-28 2,3,7,8-TCDD (TEQs)	Sample B-33 concen. (ppb)	Sample B-33 2,3,7,8-TCDD (TEQs)	Sample AP1 concen. (ppb)	Sample AP1 2,3,7,8 TCDD (TEQs)	Sample B-25* concen. (ppb)	Sample B-25* 2,3,7,8-TCDD (TEQs)	Sample B-11* concen. (ppb)	Sample B-11* 2,3,7,8-TCDD (TEQs)
		BC Grid (0 - 6")		BC Grid (0 - 10")		BC Grid (0 - 3")		BC Grid (0 - 6")		BC Grid (0 - 6")	
non 2,3,7,8-TCDFs	0	nd	-	nd	-	nd	-	nd	-	nd	-
2,3,7,8-TCDF	0.1	nd	-	nd	-	nd	-	nd	-	nd	-
non 2,3,7,8-PeCDFs	0	1.1	0	nd	-	nd	-	9.4	0	0.92	0
1,2,3,7,8-PeCDF	0.05	nd	-	nd	-	nd	-	nd	-	nd	-
2,3,4,7,8-PeCDF	0.5	nd	-	nd	-	nd	-	nd	-	nd	-
non 2,3,7,8-HxCDFs	0	49	0	110	0	92	0	247.3	0	14	0
1,2,3,4,7,8-HxCDF	0.1	2.5	0.25	nd	-	5.4	0.54	2.7	0.27	nd	-
1,2,3,6,7,8-HxCDF	0.1	0.73	0.073	nd	-	1.6	0.16	nd	-	nd	-
2,3,4,6,7,8-HxCDF	0.1	nd	-	nd	-	nd	-	nd	-	nd	-
1,2,3,7,8,9-HxCDF	0.1	1.8	0.18	nd	-	nd	-	10	1.0	nd	-
non 2,3,7,8-HpCDFs	0	419	0	477	0	599	0	1208	0	47.6	0
1,2,3,4,6,7,8-HpCDF	0.01	86	0.86	110	1.1	160	1.6	250	2.5	11	0.11
1,2,34,7,8,9-HpCDF	0.01	15	0.15	13	0.13	21	0.21	42	0.42	1.4	0.014
OCDF	0.001	3200	3.2	920	0.920	5900	5.9	2400	2.4	110	0.110
TEQs PCDFs			4.713		2.15				6.59		0.234
non 2,3,7,8-TCDDs	0	nd	-	nd	-	nd	-	nd	-	nd	-
2,3,7,8-TCDD	1	nd	-	nd	-	nd	-	nd	-	nd	-
non 2,3,7,8-PeCDDs	0	0.01	0	nd	-	0.7	0	nd	-	nd	-
1,2,3,7,8-PeCDD	0.5	0.52	0.26	nd	-	2.1	1.05	nd	-	nd	-
non 2,3,7,8-HxCDDs	0	17	0	61	0	46.2	0	108.9	0	4.8	0
1,2,3,4,7,8-HxCDD	0.1	2.5	0.25	nd	-	7.8	0.78	nd	-	nd	-
1,2,3,6,7,8-HxCDD	0.1	14	1.4	19	1.9	31	3.1	16	1.6	nd	-
1,2,3,7,8,9-HxCDD	0.1	4.6	0.46	nd	-	13	1.3	5.1	0.51	nd	-
non 2,3,7,8-HpCDDs	0	230	0	430	0	500	0	900	0	40	0
1,2,3,4,6,7,8-HpCDD	0.01	770	7.7	970	9.7	1600	16.0	2300	23	100	1.0
OCDD	0.001	5600	5.6	5700	5.7	7100	7.1	13000	13	670	0.67
Total TEQS OCDD/OCDF			8.8		6.62		13.00		15.40		0.78
TEQs NON-OCDD/OCDF			11.58		12.83		24.74		29.30		1.12
TOTAL 2,3,7,8-TCDD TEQS			**20.38**		**19.45**		**37.74**		**44.70**		**1.90**

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TABLE 7.1 (cont)

CALCULATION OF 2,3,7,8-TCDD TOXIC EQUIVALENTS

Congeners	1989 TEFs	Sample T2C3 concen. (ppb)	2,3,7,8-TCDD (TEQs)	Sample T2C5 concen. (ppb)	2,3,7,8-TCDD (TEQs)	Sample T3C3 concen. (ppb)	2,3,7,8-TCDD (TEQs)	Sample R2C2 concen. (ppb)	2,3,7,8-TCDD (TEQs)
		TC Grid (0 - 6")		TC Grid (0 - 6")		TC Grid (1.1 - 2.1")		RC Grid (1 - 9")	
non 2,3,7,8-TCDFs	0	nd	-	nd	-	nd	-	0.69	0
2,3,7,8-TCDF	0.1	nd	-	nd	-	nd	-	nd	-
non 2,3,7,8-PeCDFs	0	1.1	0	nd	-	nd	-	8.6	0
1,2,3,7,8-PeCDF	0.05	nd	-	nd	-	nd	-	nd	-
2,3,4,7,8-PeCDF	0.5	nd	-	nd	-	nd	-	nd	-
non 2,3,7,8-HxCDFs	0	52.58	0	105.3	0	14.8	0	278	0
1,2,3,4,7,8-HxCDF	0.1	2.7	0.27	4.7	0.47	1.2	0.12	12	1.2
1,2,3,6,7,8-HxCDF	0.1	0.56	0.056	nd	-	nd	-	nd	-
2,3,4,6,7,8-HxCDF	0.1	0.36	0.036	nd	-	nd	-	nd	-
1,2,3,7,8,9-HxCDF	0.1	1.8	0.18	nd	-	nd	-	nd	-
non 2,3,7,8-HpCDFs	0	386	0	487.5	0	68.9	0	1070	0
1,2,3,4,6,7,8-HpCDF	0.01	93	0.93	93	0.93	14	0.14	200	2.0
1,2,34,7,8,9-HpCDF	0.01	11	0.11	9 5	0.095	2.1	0 021	30	0.30
OCDF	0.001	2700	2.7	nd	-	150	0.150	1700	1.70
TEQs PCDFs		4.272			2.345		0.431		5.20
non 2,3,7,8-TCDDs	0	nd	-	nd	-	nd	-	nd	-
2,3,7,8-TCDD	1	nd	-	nd	-	nd	-	nd	-
non 2,3,7,8-PeCDDs	0	nd	-	nd	-	nd	-	11.3	0
1,2,3,7,8-PeCDD	0.5	nd	-	nd	-	nd	-	2.7	1.35
non 2,3,7,8-HxCDDs	0	19.1	0	20	0	2.53	0	167	0
1,2,3,4,7,8-HxCDD	0.1	2.5	0.25	4.5	0.45	nd	-	12	1.2
1,2,3,6,7,8-HxCDD	0 1	12	1.2	16	1.8	2.6	0.26	44	4.4
1,2,3,7,8,9-HxCDD	0 1	4.4	0.44	6.5	0.65	0.47	0.047	17	1.7
non 2,3,7,8-HpCDDs	0	300	0	320	0	50	0	1500	0
1,2,3,4,6,7,8 HpCDD	0.01	900	9.0	600	6.6	140	1.40	1900	19 0
OCDD	0.001	5100	5.1	4000	4.0	820	0.82	20000	20.0
Total TEQs OCDD/OCDF			7.80		4.0		0.97		21.70
TEQs NON-OCDD/OCDF			12.46		11.65		1.99		31.15
TOTAL 2,3,7,8-TEQS			**20.26**		**15.65**		**2.96**		**52.85**

008817

TABLE 7.1 (cont)
CALCULATION OF 2,3,7,8-TCDD TOXIC EQUIVALENTS

Congeners	1989 TEFs	Sample R2C3 concen. (ppb)	Sample R2C3 2,3,7,8-TCDD (TEQs)	Sample R2C4 concen. (ppb)	Sample R2C4 2,3,7,8-TCDD (TEQs)	Sample R3C3 concen. (ppb)	Sample R3C3 2,3,7,8-TCDD (TEQs)
		RC Grid (1 - 2.5")		RC Grid (0 - 1.5")		RC Grid (0 - 6)	
non 2,3,7,8-TCDFs	0	nd	-	0.29	0	nd	-
2,3,7,8-TCDF	0.1	nd	-	nd	-	nd	-
non 2,3,7,8-PeCDFs	0	5.6	0	2.2	0	nd	
1,2,3,7,8-PeCDF	0.05	nd	-	nd	-	nd	-
2,3,4,7,8-PeCDF	0.5	nd	-	nd	-	nd	-
non 2,3,7,8-HxCDFs	0	202	0	101.9	0	340	0
1,2,3,4,7,8-HxCDF	0.1	18	1.8	8.1	0.81	nd	-
1,2,3,6,7,8-HxCDF	0.1	nd	-	nd	-	nd	-
2,3,4,6,7,8-HxCDF	0.1	nd	-	nd	-	nd	-
1,2,3,7,8,9-HxCDF	0.1	nd	-	nd	-	nd	-
non 2,3,7,8-HpCDFs	0	1294	0	569	0	2225	0
1,2,3,4,6,7,8-HpCDF	0.01	260	2.60	120	1.20	420	4.20
1,2,3,4,7,8,9-HpCDF	0.01	46	0.46	21	0.21	55	0.55
OCDF	0.001	7200	7.20	2300	2.30	5100	5.10
TEQs PCDFs			12.06		4.52		9.85
non 2,3,7,8-TCDDs	0	nd	-	4.3	0	nd	-
2,3,7,8-TCDD	1	nd	-	nd	-	nd	-
non 2,3,7,8-PeCDDs	0	1.3	0	28.7	0	14	0
1,2,3,7,8-PeCDD	0.5	3.7	1.85	2.3	1.15	nd	-
non 2,3,7,8-HxCDDs	0	89	0	67	0	293	0
1,2,3,4,7,8-HxCDD	0.1	22	2.2	10	1.0	24	2.4
1,2,3,6,7,8-HxCDD	0.1	67	6.7	29	2.9	90	9.0
1,2,3,7,8,9-HxCDD	0.1	42	4.2	14	1.4	33	3.3
non 2,3,7,8-HpCDDs	0	1600	0	800	0	3000	0
1,2,3,4,6,7,8 HpCDD	0.01	3200	32	1300	13.0	5000	50.0
OCDD	0.001	23000	23	9000	9.00	46000	46.0
Total TEQs OCDD/OCDF			30.2		11.30		51.10
TEQs NON-OCDD/OCDF			51.81		21.67		69.45
TOTAL 2,3,7,8-TCDD TEQS			**82.01**		**32.97**		**120.55**

008818

(4) preclude the demonstration of a lognormal data set for these sample grids.

The application of the W test to the distribution of the PCDD/PCDF data for the B grid indicate that the dioxin concentrations are normally distributed for this data set. The W test is a rigorous statistical analysis which tests whether a data set has been drawn from an underlying normal distribution (Gilbert, 1987). Normal distribution of soil sample data from the B grid was further confirmed by plotting cumulative frequencies versus total TCDD equivalent concentrations. These plots confirmed linearity of the non-transformed data (Appendix B). Accordingly, for the purpose of this review, the arithmetic mean concentrations below were used as representative contaminant concentrations.

	ppb		
	Grid B	Grid TC	Grid RC
OCDD	8.0	4.2	28.5
Non-OCDD	15.4	8.7	43.5

As in the 1989 EA, only the surficial samples used to calculate these concentrations. This is appropriate since human contact with soil is usually limited to the top 0-3 inches.

7.2 Sample Grids

In the 1989 EA, the TC and B grids were assumed to contribute equally to the mean concentration at the Main Site. Since the TC grid comprises only 1.3% of the area of the main site, it seems reasonable to weight the average concentrations of the B and TC grids accordingly to derive a representative contaminant concentration for the main site. Specifically, for the purposes of assessing health risks at the main site, the representative concentrations for the main site will be the sum of 1.3% of the mean concentration of the TC grid and 98.7% of the mean concentration of the B grid.

7.3 Time-Dependent Contaminant Concentrations

The half life of PCDD/PCDFs in soil was considered in this review. A soil half life of 16 years was assumed (Kimbrough et al., 1984). The decay rate constant for a half life of 16 years is 0.043 $yr.^{-1}$, which represents the constant fraction degraded per year. Representative soil concentrations for each age group over a 70 year lifetime were derived using the following equation:

$$-\frac{dN}{dt} = kN$$

Where:

- dN = the amount of chemical decaying per unit time
- dt = increment of time
- k = the decay constant (fraction degraded per time)
  (k = 0.693/half life for first order reactions)
  half life
- N = the total amount of chemical present at any given time

The integrated form of this equation was used to calculate the amount of contaminant remaining after an average time period for a lifetime (0-70 years) exposure for each age group exposed.

Integrated equation:

$$\ln \frac{N_o}{N} = kt$$

or

$$2.3 \log \frac{N^o}{N} = kt$$

Where;

N^o = amount of chemical at t=o (100%)

N = percent remaining at time t

t = average age of soil

The concentration of OCDD/OCDF isomers and non-OCDD/OCDF isomers used to calculate health risks are summarized below.

ppb

	Railroad Ditch		Main Site	
	OCDD	Non-OCDD	OCDD	Non-OCDD
Child (6-12 yrs)	19.4	29.5	5.43	10.5
Adult (12-70 yrs)	4.90	7.44	1.37	2.63
Railroad personnel	15.0	22.8	...	...

8.0 DOSE CALCULATION AND HEALTH RISK ESTIMATES

This section describes the methodologies used to quantitate contaminant uptake (dose) and associated health risks.

8.1 Exposure Scenarios

As described in the 1989 EA, Exposure Scenario II represents the most probable future land use conditions which assume unrestricted access to the Main Site as well as the Railroad Ditch. As in the 1989 EA, this review evaluates risks to: 1) children and adults due to exposure at the main site, and 2) risks to railroad personnel, children, and adults due to exposure at the ditch. The same age groups used in the 1989 EA (6-12 years and 12-70 years) are used in this review. All exposure assumptions used in the 1989 EA are reproduced here, except for the refinements presented in Table 6-1. The main assumptions used in the 1989 EA that are reproduced here are as follows:

- No exposure will occur for the 0-6 year age group.
- Children of age 6-12 years will visit the railroad ditch and main site six-times a year.
- Adults (age 12-70 years) will visit the railroad ditch and main site twelve times a year.
- railroad personnel will come in contact with the railroad ditch six times a year for 30 years.

8.2 General Intake Equation

The total lifetime average daily dose (LADD) is used to quantitatively estimate potential cancer risks. The total LADD is the sum of the LADDs

008822

calculated for each age group. Oral and dermal LADDs for each age group are calculated according to the following general equation:

$$I = \frac{C \times CR \times EFD}{BW \times AT}$$

Where;

I = intake; the amount of chemical (mg/kg body weight-day) at the exchange boundary (e.g., skin or gastrointestinal tract)

Chemical-related variable

C = chemical concentration; the average concentration contacted over the exposure period (e.g., mg/kg soil)

Variables that describe the exposed population

CR = contact rate; the amount of contaminated medium contacted per unit time or event (e.g., kg soil/day)

EFD = exposure frequency and duration; describes how long and how often exposure occurs. Often calculated using two terms (EF and ED):

EF = exposure frequency (days/year)

ED = exposure duration (years)

BW = body weight; the average body weight over the exposure period (kg)

AT = averaging time; period over which exposure is averaged (e.g., years)

This general equation is consistent with the general dose equation presented in the USEPA'S 1989 Risk Assessment Guidance for Superfund and is consistent with the equation described in the 1989 EA.

As described in USEPA's 1989 Risk Assessment Guidance for Superfund, it is critical to normalize the estimated total dose of a carcinogen over a lifetime of exposure. For example, in order to estimate the potential cancer risk associated with six years of exposure to a carcinogen in soil, the total dose absorbed during those six years must be divided by a 70 year lifetime. Cancer risks are assessed in this manner because tumor development is typically a lifetime process.

8.3 Specific Intake Equations

The general intake equation is modified for soil ingestion and dermal absorption as follows:

$$\underline{\text{Ingestion Intake}}\ (\text{mg/kg-day}) = \frac{CS \times IR \times CF \times FI \times ABS \times EF \times ED}{BW \times AT}$$

Where:

CS = Chemical Concentration in Soil (mg/kg)

IR = Ingestion Rate (mg soil/day)

CF = Conversion Factor (10^{-6} kg/mg)

FI = Fraction Ingested from Contaminated Source (unitless)

ABS = Absorption Factor (unitless)

EF = Exposure Frequency (days/years)

ED = Exposure Duration (years)

BW = Body Weight (kg)

008824

AT = Averaging Time (period over which exposure is averaged-days)

$$\text{Absorbed Dermal Dose (mg/kg-day)} = \frac{CS \times CF \times SA \times AF \times ABS \times EF \times ED}{BW \times AT}$$

Where:

CS = Chemical Concentration in Soil (mg/kg)

CF = Conversion Factor (10^{-6} kg/mg)

SA = Skin Surface Area Available for Contact ($cm^2$3/event)

AF = Soil to Skin Adherence Factor (mg/cm^2)

ABS = Absorption Factor (unitless)

EF = Exposure Frequency (events/year)

ED = Exposure Duration (years)

BW = Body Weight (kg)

AT = Averaging Time (period over which exposure is averaged)

8.4 Calculation of Total LADDs and Cancer Risks

Refined exposure assumptions used to estimate LADDs are listed in Table 6-1. All other exposure assumptions are consistent with the 1989 EA. The spreadsheet calculations used to derive the LADDs are provided in Appendix A. The total LADDs and cancer risks are summarized below. The cancer risks are simply the product of the total LADD (mg/kg/day) and the cancer potency factor $(9{,}700\ mg/kg/day)^{-1}$.

In summary, potential increased cancer risk at the main site and the railroad ditch are on the order of 10^{-8}. These risk values are approximately 2-3 orders of magnitude lower than those derived for the

	Railroad Ditch		Main Site
	Railroad Personnel	Regular Visitor	Regular Visitors
Route Specific LADD			
Oral	6.5×10^{-13} mg/kg/day	4.7×10^{-12} mg/kg/day	1.7×10^{-12} mg/kg/day
Dermal	7.3×10^{-13} mg/kg/day	1.2×10^{-12} mg/kg/day	3.8×10^{-13} mg/kg/day
Total LADD	1.4×10^{-12}	5.9×10^{-12}	2.1×10^{-12}

same scenario (Scenario II) in the EA (7×10^{-6} - 2×10^{-5} for the main site and the ditch, respectively). The lower risk estimates obtained in this review are a result of the use of refined exposure assumptions taken from the scientific literature. The refinements primarily responsible for the lower health risks estimates are:

- Use of more accurate soil ingestion rates, and
- Use of specific bioavailability estimates for OCDD.

Accordingly, the health risk estimates presented in the 1989 EA should be considered upper-bound estimates of potential cancer risk.

9.0 RISK CHARACTERIZATION

This section discusses the magnitude of the health risk estimates derived in Section 8.0 and places them in perspective with respect to risk levels that are typically considered acceptable by the regulatory and public community.

9.1 Characterization of Non-Cancer Hazards

As described in Section 5.1, several regulatory agencies have established daily acceptable daily intake levels (ADIs) of dioxin. These ADIs range from 1-10 pg/Kg-day and are considered to be protective of non-cancer effects. The total LADDs determined in this evaluation for the Arkwood site are several orders of magnitude below the ADIs, and therefore, it can be calculated that the non-cancer risks at the Arkwood site are negligible.

9.2 Characterization of Cancer Risks

As described in Section 8.0, potential dioxin cancer risks at Arkwood are on the order of 10^{-8}. Beginning in the late 1970's and early 1980's, regulatory agencies in the U.S. and abroad frequently adopted a cancer risk criteria of one in a million as a negligible (i.e., of no concern) risk when fairly large populations might be exposed to a suspect carcinogen. Unfortunately, theoretical increased cancer risks of one in a million are often incorrectly portrayed as serious public health risks. As recently discussed by Dr. Frank Young, (FDA, 1988) the current commissioner of the FDA, this was not the intent of such estimates:

> In applying the de minimis concept and in setting other safety standards, FDA has been guided by the figure of "one in a million." Other Federal agencies have also used a one in a million level, such as the Occupational Safety and Health

> Administration and the Environmental Protection Agency. Both agencies rely on the one in one million increased risk over a lifetime as a reasonable criterion for separating high-risk problems warranting agency attention from negligible risk problems that do not.
>
> The risk level of one in one million is often misunderstood by the public and the media. It is not an actual risk - i.e., we do not expect one out of every million people to get cancer if they drink decaffeinated coffee. Rather, it is a mathematical risk based on scientific assumptions used in risk assessment. FDA uses a conservative estimate to ensure that the risk is not understated. We interpret animal test results conservatively and we are extremely careful when we extrapolate risks to humans. When FDA uses the risk level of one in one million, it is confident that the risk to humans is virtually nonexistent.

In short, a 'one in a million' cancer risk estimate, which is often tacitly assumed by some policy-makers to represent a trigger level for regulatory action, actually represents a level of risk that is so small as to be of negligible concern.

Another misperception within the risk assessment arena is that all occupational and environmental regulations have as their goal a theoretical maximum cancer risk of 1 in 1,000,000. Travis et al. (1987) recently conducted a retrospective examination of the level of risk which triggered regulatory action in 132 decisions. Three variables were considered: 1) individual risk (an upper-limit estimate of the probability that the most highly exposed individual in a population will develop cancer as a result of a lifetime exposure), 2) population risk (an upper-limit estimate of the number of additional incidences of cancer in the

exposed population), and 3) population size. The findings of Travis et al. (1987) can be summarized as follows:

1. Every chemical with an individual lifetime risk above 4×10^{-3} received regulation. Those with values below 1×10^{-6} remained unregulated.

2. For small populations, regulatory action never resulted for individual risks below 1×10^{-4}.

3. For effects resulting from exposures to the entire U.S. population, a risk level below 1×10^{-6} never triggered action; above 3×10^{-4} it always triggered action.

In short, regulatory agencies have found risks far in excess of 1 in 1,000,000 acceptable if experienced by small populations. Not only have regulatory agencies taken exception to the unilateral application of 1 in 1,000,000 risk, but many common human activities entail risks greatly in excess of 1 in 1,000,000. Rodricks et al. (1987) has discussed these:

> Examination of the risks of common human activities demonstrates....a lifetime risk of 1 in 100,000 or more is within the realm of, or orders of magnitude below, everyday risks that generally do not cause undue concern. These are risks that people, while they are aware of them and may have some concern or fear over them, do not in general alter their behavior to avoid. The risks from many activities greatly exceed the level of one in 100,000. In comparison to these background risks of "everyday activities," a lifetime risk of 1 in 100,000 is relatively small. Accordingly, regulatory action will not generally be justifiable unless risks are substantially higher than this 1 in 100,000 "benchmark".

In essence, society attempts to reduce the risks associated with exposure to chemicals to levels much lower than those to which we voluntarily expose ourselves each day; such as driving a car, smoking, using artificial sweeteners, and travelling in commercial aircraft. The key issue is one of involuntary vs. voluntary risk.

As discussed above, U.S. Federal regulatory agencies have adopted a 'one in a million' cancer risk as being of negligible concern in situations where large populations (e.g., 200 million people) are involuntarily exposed to suspect carcinogens (e.g., food additives). When smaller populations are exposed (e.g., in occupational settings) theoretical cancer risks of up to 10^{-4} have been considered acceptable. In short, an estimated cancer risk of less than one in a million for a population of less than 2,000 individuals is far below the maximal risk levels historically considered to be protective of public health.

It is important to emphasize that an estimated cancer risk of one in a million (or less) does not actually imply that an additional one out of every million people will get cancer. Rather, the 'one in a million' value is simply a mathematical estimate, conservatively derived from animal exposure studies, that has been historically interpreted as representing a nonexistent risk.

9.3 Background Levels of Dioxin Uptake and Body Burden

Longstreth and Hushon (1985) have estimated that the general population has an average daily intake of 7 fg/kg-day of 2,3,7,8-TCDD. This background intake is primarily the result of low 2,3,7,8-TCDD levels in the food chain. As shown in Section 8.4, the highest estimated daily dioxin intake at the Arkwood site is 5.8×10^{-12} mg/kg-day, or 5.8 fg/kg-day. Hence, the levels of dioxin uptake at Arkwood are approximately the same as the negligible levels experienced by the rest of the U.S. population.

It has been demonstrated that typical body burdens of OCDD in human adipose range from 600-800 ppt. As described below a 70-year exposure to the average OCDD concentration in the B grid (8.0 ppb) would yield a total OCDD dose of 2.6×10^{-3} mg, according to the exposure assumptions used in this evaluation:

Oral

6-12 yrs: 5 mg soil/event x 6 events/yr x 6 years x 0.01 x 8 µgOCDD/kg soil x 1 kg/10^6 mg = 1.4×10^{-5} mg

12-70 yrs: 5 mg soil/event x 12 events/yr x 58 years x .01 x 8 μgOCDD/Kg soil x 1 Kg/10^6 mg = 2.8 x 10^{-4} mg

Total Oral = 2.9 x 10^{-4} mg OCDD

Dermal

6-12 yrs: 0.5 mg soil/cm^2 skin x 625 cm^2 skin/event x 6 events/yr x .001 x 8 μgOCDD/Kg soil x 1 Kg/10^6 mg = 9 x 10^{-5} mg OCDD

12-70 yrs: 0.5 mg soil/cm^2 skin x 840 cm^2 skin/event x 12 events/year x 58 years x .001 x 8 μgOCDD/Kg soil x 1 Kg/10^6 mg = 2.3 x 10^{-3} mg

Total Dermal = 2.4 x 10^{-3} mg

Total oral and dermal uptake = 2.9 x 10^{-4} mg + 2.4 x 10^{-3} mg = 2.6 x 10^{-3} mg

The average human adult contains 21 kg of body fat (Snyder, 1975). If all of the OCDD absorbed from the main site over a 70-year lifetime were to be distributed evenly throughout the adipose, the resulting concentration would be 2.6 x 10^{-6} Kg OCDD/21 Kg fat = 123 ppt.

This is an extremely conservative estimate of the OCDD adipose burden associated with exposure to the main site because it assumes that 1) all absorbed OCDD deposits in adipose, even though it is known that a significant fraction of absorbed OCDD deposits in other tissues and 2) the absorbed OCDD is never eliminated, even though OCDD has a biological half-life of 3-5 months.

Consequently it is reasonable to assume that the levels of OCDD that would accumulate in humans who visited the main site at Arkwood on a regular basis would be negligible compared to the pre-existing background levels.

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APPENDIX A

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ORAL main site spreadsheet

	A	B	C
1	CONGENER	RECEPTOR POPULATION	SOIL INGESTED
2			
3			(mg / exposure event)
4	NON OCTA		
5		CHILD	5.00
6			
7		ADULT	5.00
8			
9			
10	OCDD/OCDF		
11		CHILD	5.00
12			
13		ADULT	5.00
14			
15			
16			
17			
18			
19			
20			
21			
22			

008835

ORAL main site spreadsheet

	D	E	F
1	EXPOSURE FREQUENCY	CONVERSION FACTOR	BIOAVAILABILITY
2			
3	(exposure events/year)	(kg/mg)	
4			
5	6.00	1.00E-06	0.50
6			
7	12.00	1.00E-06	0.50
8			
9			
10			
11	6.00	1.00E-06	0.01
12			
13	12.00	1.00E-06	0.01
14			
15			
16			
17			
18			
19			
20			
21			
22			

008836

ORAL main site spreadsheet

	G	H	I	J
1	EXPOSURE	TIME CONVERSION	1/BODY WEIGHT	1/AVERAGING
2	DURATION	FACTOR		TIME
3	(years)	(years/day)	(1/kg)	(1/years)
4				
5	6.00	2.74E-03	3.45E-02	1.43E-02
6				
7	58.00	2.74E-03	1.43E-02	1.43E-02
8				
9				
10				
11	6.00	2.74E-03	3.45E-02	1.43E-02
12				
13	58.00	2.74E-03	1.43E-02	1.43E-02
14				
15				
16				
17				
18				
19				
20				
21				
22				

008837

ORAL main site spreadsheet

	K	L
1	ARITHMETIC MEAN	LADD
2	SOIL CONCENTRATION	(mg/kg - day)
3	(mg/kg)	
4		
5	1.05E-02	1.49E-04
6		
7	2.63E-03	3.76E-05
8		
9		
10		
11	5.45E-03	7.79E-05
12		
13	1.37E-03	1.96E-05
14		
15		
16	ORAL LADD	3.9 x 10-12 mg/kg/day
17	for a 70-year	
18	exposure	
19		
20	ORAL LADD	1.7 x 10-12 mg/kg/day
21	for a 30-year	
22	exposure duration	

008838

	A	B	C	D	E	F	G
1	CONGENER	RECEPTOR POPULATION	SOIL INGESTED	EXPOSURE FREQUENCY	CONVERSION FACTOR	BIOAVAILABILITY	EXPOSURE DURATION
2							
3			(mg / exposure event)	(exposure events/year)	(kg/mg)		(years)
4	NON OCTA						
5		CHILD	5 00	6 00	1 00E 06	0 50	6 00
6							
7		ADULT	5 00	12 00	1 00E 06	0 50	58 00
8							
9		RR PERSONNEL	5 00	6 00	1 00E 06	0 50	30 00
10							
11	OCDD/OCDF						
12		CHILD	5.00	6.00	1 00E 06	0 01	6.00
13							
14		ADULT	5 00	12.00	1 00E 06	0 01	58 00
15							
16		RR PERSONNEL	5.00	6 00	1 00E 06	0 01	30 00
17							
18							
19							
20							
21							
22							
23							
24							
25							
26							
27							
28							
29							
30							

008839

	H	I	J	K	L	M
1	TIME CONVERSION	1/BODY WEIGHT	1/AVERAGING	ARITHMETIC MEAN	LADD	TOTAL NORMALIZED
2	FACTOR		TIME	SOIL CONCENTRATION	(mg/kg day)	LADD
3	(years/day)	(1/kg)	(1/years)	(mg/kg)		(mg/kg day)
4						
5	2 74E-03	3 45E-02	1 43E-02	2 95E-02	4 22E-04	1 81E-04
6						
7	2 74E-03	1 43E-02	1 43E-02	7 44E-03	1 06E-04	4 56E-05
8						
9	2 74E-03	1 43E-02	1 43E-02	2 28E-03	3 26E-05	5 75E-13
10						
11						
12	2 74E-03	3 45E-02	1 43E-02	1 94E-02	2 77E-04	1 19E-04
13						
14	2 74E-03	1 43E-02	1 43E-02	4 90E-03	7 01E-05	3 01E-05
15						
16	2 74E-03	1 43E-02	1 43E-02	1 50E-02	2 15E-04	7 56E-14
17						
18						
19				ORAL LADD	6.5 X 10-13 mg/kg/day	
20				for railroad personnel		
21						
22				ORAL LADD	1 0 x 10-11 mg/kg/day	
23				for regular visitors,		
24				70-year exposure		
25				duration		
26						
27				ORAL LADD	4.7 x 10-12 mg/kg/day	
28				for regular visitors,		
29				adjusted for a 30-year		
30				exposure duration		

008840

	A	B	C	D	E	F	G
1	CONGENER	RECEPTOR POPULATION	SKIN SURFACE AREA (cm2/event)	AMOUNT OF SOIL ADHERING TO SKIN (mg/cm2)	EXPOSURE FREQUENCY (exposure events/year)	CONVERSION FACTOR (kg/mg)	BIOAVAILABILITY
2							
3							
4	NON OCTA						
5		CHILD	625 00	0 50	6 00	1 00E 06	0 001
6							
7		ADULT	840 00	0 50	12 00	1 00E 06	0 001
8							
9		RR PERSONNEL	840 00	0 50	6 00	1 00E 06	0 C01
10							
11	OCDD/OCDF						
12		CHILD	625 00	0 50	6 00	1 00E 06	0 001
13							
14		ADULT	840 00	0 50	12 00	1 00E 06	0 001
15							
16		RR PERSONNEL	840 00	0 50	6 00	1 00E 06	0 001
17							
18							
19							
20							
21							
22							
23							
24							
25							
26							
27							
28							
29							
30							

008841

	H	I	J	K	L	M
1	EXPOSURE	TIME CONVERSION	1/BODY WEIGHT	1/AVERAGING	ARITHMETIC MEAN	LADD
2	DURATION	FACTOR		TIME	SOIL CONCENTRATION	(mg/kg day)
3	(years)	(years/day)	(1/kg)	(1/years)	(mg/kg)	
4						
5	6.00	2.74E-03	3.45E-02	1.43E-02	2.95E-02	4.22E-04
6						
7	58.00	2.74E-03	1.43E-02	1.43E-02	7.44E-03	1.06E-04
8						
9	30.00	2.74E-03	1.43E-02	1.43E-02	2.28E-03	3.26E-05
10						
11						
12	6.00	2.74E-03	3.45E-02	1.43E-02	1.94E-02	2.77E-04
13						
14	58.00	2.74E-03	1.43E-02	1.43E-02	4.90E-03	7.01E-05
15						
16	30.00	2.74E-03	1.43E-02	1.43E-02	1.50E-02	2.15E-04
17						
18						
19					DERMAL LADD	7.3 x 10-13 mg/kg/day
20					for railroad personnel	
21						
22					DERMAL LADD	2.8 x 10-12 mg/kg/day
23					for regular visitors,	
24					70-year exposure	
25					duration	
26						
27					DERMAL LADD	1.2 x 10-12 mg/kg/day
28					for regular visitors,	
29					adjusted for a 30-year	
30					exposure duration	

008842

APPENDIX B

008843

APPENDIX B

THE SHAPIRO-WILK TEST FOR NORMALITY

$d = \Sigma (x_i - \bar{x})^2 = \Sigma x_i^2 - 1/n (\Sigma xi)^2$

$\bar{x} = 23.55$

		x	$(xi_i - \bar{x})_2$	$(xi_i)^2$
i=	1	39.43	252.17	1554.72
	2	37.74	201.36	1424.31
	3	27.80	18.06	77: 84
	4	20.38	10.05	415.34
	5	19.45	16.81	378.30
	6	15.18	70.06	230.43
	7	4.91	347.45	24.11
	Σx =	164.89	Σ=915.96 (-d)	Σ= 4800.05

$4800.05 - [1/7 (27188.71)] = 915.95 = d$

$d = 915.96$
$n = 7$
$k = \frac{n-1}{2} = 3$

find coefficients from Table A6:

$a_1 = 0.6233$
$a_2 = 0.3031$
$a_3 = 0.1401$

$$w = \frac{1}{d} [\Sigma a_i (x_{(n-i+1)} - x_i)]^2$$

$$w = \frac{1}{915.95} ([(0.6233(4.91 - 39.43)] + [0.3031 (15.18 - 37.74)]$$

$$+ [0.1401 (19.45 - 27.80)])^2$$

$$= 1.090E-03 [(-21.52) + (-6.84) + (-1.17)]^2$$

$$= 9.50E-01 > w_{.05} = 0.803$$

(from Table A7: Quantities of the Shapiro-Wilk Test for Normality; values of W such that 100% of the distribution of W is less than Wp)

$W > W.05$, therefore cannot reject H_o and must conclude the distribution is normal.

(H_o: The population has a normal distribution)

008844

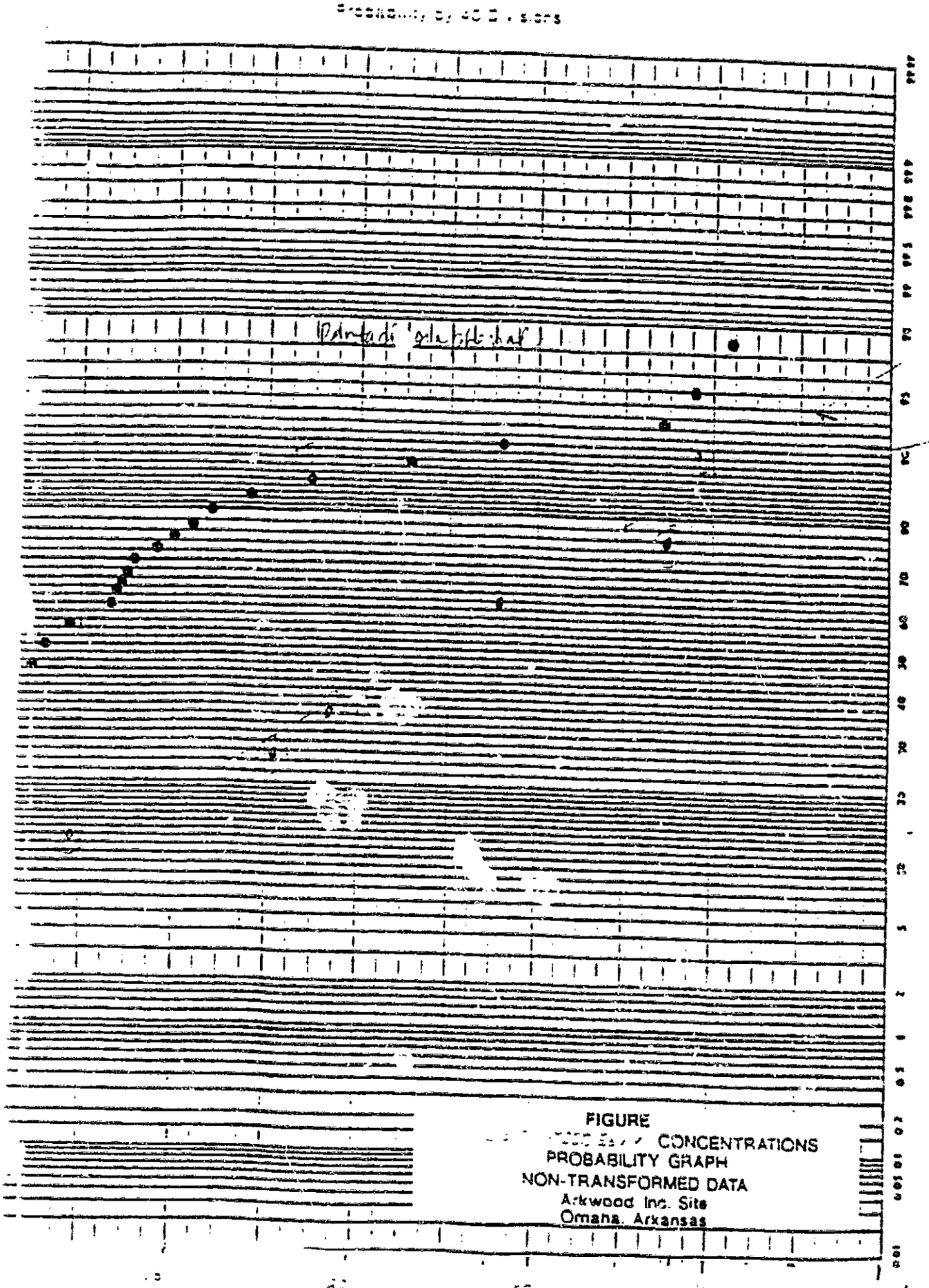

FIGURE
CONCENTRATIONS
PROBABILITY GRAPH
NON-TRANSFORMED DATA
Arkwood Inc. Site
Omaha, Arkansas

ppb

008845

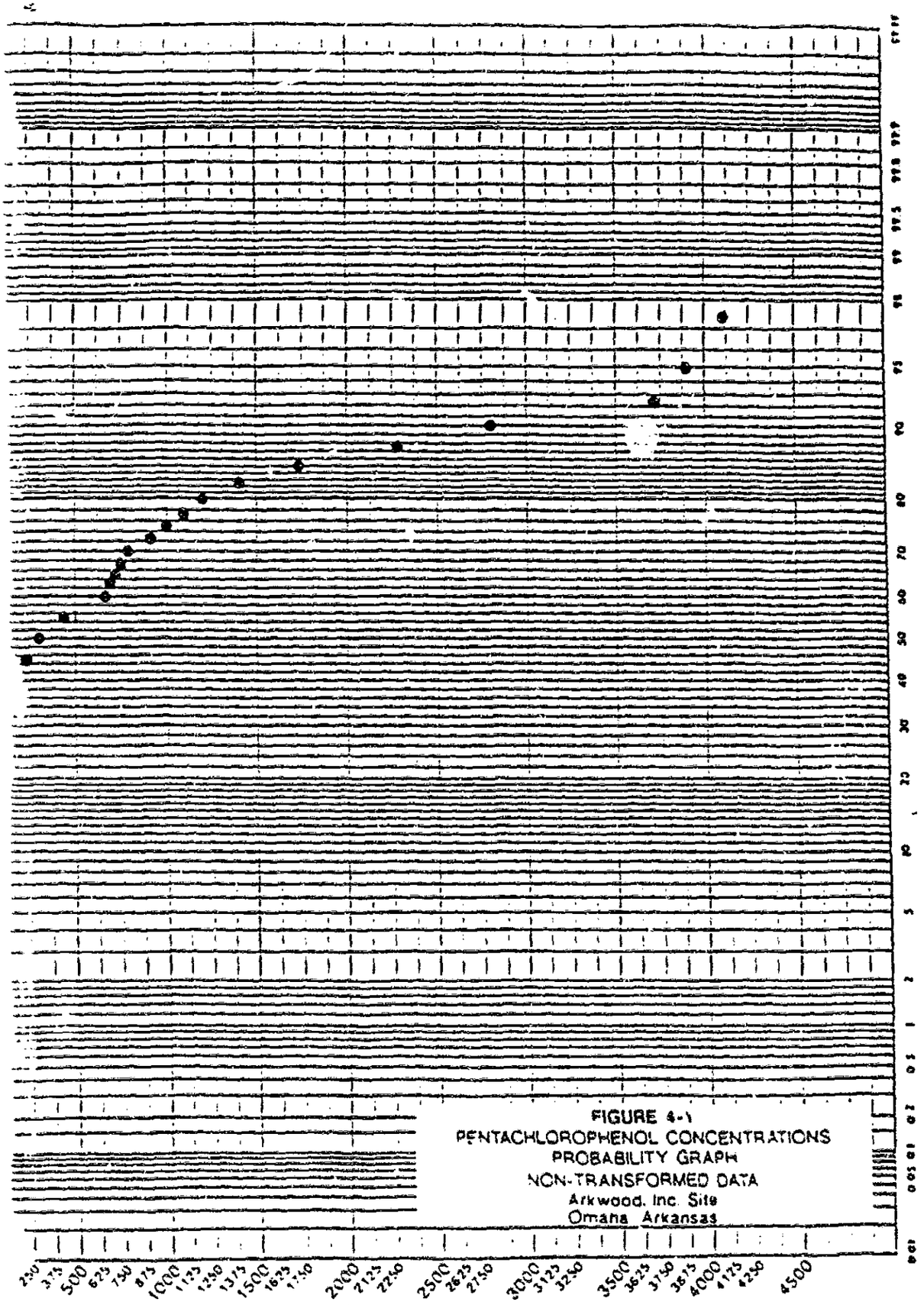

FIGURE 4-1
PENTACHLOROPHENOL CONCENTRATIONS
PROBABILITY GRAPH
NON-TRANSFORMED DATA
Arkwood, Inc. Site
Omana Arkansas

008846

Table A6 Coefficients a_i for the Shapiro-Wilk W Test for Normality

i \ n	2	3	4	5	6	7	8	9	10
1	0.7071	0.7071	0.6872	0.6646	0.6431	0.6233	0.6052	0.5888	0.5739
2	-	0.0000	0.1677	0.2413	0.2806	0.3031	0.3164	0.3244	0.3291
3	-	-	-	0.0000	0.0875	0.1401	0.1743	0.1976	0.2141
4	-	-	-	-	-	0.0000	0.0561	0.0947	0.1224
5	-	-	-	-	-	-	-	0.0000	0.0399

i \ n	11	12	13	14	15	16	17	18	19	20
1	0.5601	0.5475	0.5359	0.5251	0.5150	0.5056	0.4968	0.4886	0.4808	0.4734
2	0.3315	0.3325	0.3325	0.3318	0.3306	0.3290	0.3273	0.3253	0.3232	0.3211
3	0.2260	0.2347	0.2412	0.2460	0.2495	0.2521	0.2540	0.2553	0.2561	0.2565
4	0.1429	0.1586	0.1707	0.1802	0.1878	0.1939	0.1988	0.2027	0.2059	0.2085
5	0.0695	0.0922	0.1099	0.1240	0.1353	0.1447	0.1524	0.1587	0.1641	0.1686
6	0.0000	0.0303	0.0539	0.0727	0.0880	0.1005	0.1109	0.1197	0.1271	0.1334
7	-	-	0.0000	0.0240	0.0433	0.0593	0.0725	0.0837	0.0932	0.1013
8	-	-	-	-	0.0000	0.0196	0.0359	0.0496	0.0612	0.0711
9	-	-	-	-	-	-	0.0000	0.0163	0.0303	0.0422
10	-	-	-	-	-	-	-	-	0.0000	0.0140

i \ n	21	22	23	24	25	26	27	28	29	30
1	0.4643	0.4590	0.4542	0.4493	0.4450	0.4407	0.4366	0.4328	0.4291	0.4254
2	0.3185	0.3156	0.3126	0.3098	0.3069	0.3043	0.3018	0.2992	0.2968	0.2944
3	0.2578	0.2571	0.2563	0.2554	0.2543	0.2533	0.2522	0.2510	0.2499	0.2487
4	0.2119	0.2131	0.2139	0.2145	0.2148	0.2151	0.2152	0.2151	0.2150	0.2148
5	0.1736	0.1764	0.1787	0.1807	0.1822	0.1836	0.1848	0.1857	0.1864	0.1870
6	0.1399	0.1443	0.1480	0.1512	0.1539	0.1563	0.1584	0.1601	0.1616	0.1630
7	0.1092	0.1150	0.1201	0.1245	0.1283	0.1316	0.1346	0.1372	0.1395	0.1415
8	0.0804	0.0878	0.0941	0.0997	0.1046	0.1089	0.1128	0.1162	0.1192	0.1219
9	0.0530	0.0618	0.0696	0.0764	0.0823	0.0876	0.0923	0.0965	0.1002	0.1036
10	0.0263	0.0368	0.0459	0.0539	0.0610	0.0672	0.0728	0.0778	0.0822	0.0862
11	0.0000	0.0122	0.0228	0.0321	0.0403	0.0476	0.0540	0.0598	0.0650	0.0697
12	-	-	0.0000	0.0107	0.0200	0.0284	0.0358	0.0424	0.0483	0.0537
13	-	-	-	-	0.0000	0.0094	0.0178	0.0253	0.0320	0.0381
14	-	-	-	-	-	-	0.0000	0.0084	0.0159	0.0227
15	-	-	-	-	-	-	-	-	0.0000	0.0076

008847

A7 Quantiles of the Shapiro-Wilk W Test for Normality (Values of W_α Such That Pr(W ≤ W_α) = α)

n	$W_{0.01}$	$W_{0.02}$	$W_{0.05}$	$W_{0.10}$	$W_{0.50}$
3	0.753	0.756	0.767	0.789	0.959
4	0.687	0.707	0.748	0.792	0.935
5	0.686	0.715	0.762	0.806	0.927
6	0.713	0.743	0.788	0.826	0.927
7	0.730	0.760	0.803	0.838	0.928
8	0.749	0.778	0.818	0.851	0.932
9	0.764	0.791	0.829	0.859	0.935
10	0.781	0.806	0.842	0.869	0.938
11	0.792	0.817	0.850	0.876	0.940
12	0.805	0.828	0.859	0.883	0.943
13	0.814	0.837	0.866	0.889	0.945
14	0.825	0.846	0.874	0.895	0.947
15	0.835	0.855	0.881	0.901	0.950
16	0.844	0.863	0.887	0.906	0.952
17	0.851	0.869	0.892	0.910	0.954
18	0.858	0.874	0.897	0.914	0.956
19	0.863	0.879	0.901	0.917	0.957
20	0.868	0.884	0.905	0.920	0.959
21	0.873	0.888	0.908	0.923	0.960
22	0.878	0.892	0.911	0.926	0.961
23	0.881	0.895	0.914	0.928	0.962
24	0.884	0.898	0.916	0.930	0.963
25	0.888	0.901	0.918	0.931	0.964
26	0.891	0.904	0.920	0.933	0.965
27	0.894	0.906	0.923	0.935	0.965
28	0.896	0.908	0.924	0.936	0.966
29	0.898	0.910	0.926	0.937	0.966
30	0.900	0.912	0.927	0.939	0.967
31	0.902	0.914	0.929	0.940	0.967
32	0.904	0.915	0.930	0.941	0.968
33	0.906	0.917	0.931	0.942	0.968
34	0.908	0.919	0.933	0.943	0.969
35	0.910	0.920	0.934	0.944	0.969
36	0.912	0.922	0.935	0.945	0.970
37	0.914	0.924	0.936	0.946	0.970
38	0.916	0.925	0.938	0.947	0.971
39	0.917	0.927	0.939	0.948	0.971
40	0.919	0.928	0.940	0.949	0.972
41	0.920	0.929	0.941	0.950	0.972
42	0.922	0.930	0.942	0.951	0.972
43	0.923	0.932	0.943	0.951	0.973
44	0.924	0.933	0.944	0.952	0.973
45	0.926	0.934	0.945	0.953	0.973
46	0.927	0.935	0.945	0.953	0.974
47	0.928	0.936	0.946	0.954	0.974
48	0.929	0.937	0.947	0.954	0.974
49	0.929	0.937	0.947	0.955	0.974
50	0.930	0.938	0.947	0.955	0.974

Source: After Shapiro and Wilk, 1965

The null hypothesis of a normal distribution is rejected at the α significance level if the calculated W is less than

This table is used in Section 12.3.1

008848

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APPENDIX B

APPENDIX B

Review and Critique of the Scientific Basis of the TEF for OCDD

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EXECUTIVE SUMMARY

This document provides a detailed review and commentary on the scientific basis of the 1989 toxic equivalency factor (TEF) for occachlorodibenzo-p-dioxin (OCDD). In 1989, the USEPA revised the TEF from zero to 0.001 based on a study which demonstrated that the OCDD concentration in the liver required to maximally induce liver enzymes is 1,000 times the required 2,3,7,8-TCDD concentration. The new TEF of 0.001 indicates that the USEPA will treat OCDD as a carcinogen with 1/1000 the carcinogenic potential of 2,3,7,8-TCDD. The main conclusions of this review are as follows:

- The TEF system assumes that non-cancer effects such as liver enzyme induction can be used quantitatively to estimate carcinogenic potential. However, for the dioxin isomers, there is no clear correlation between increased non-cancer effects and carcinogenic potential. Consequently, dioxin isomers with no carcinogenic potential might be assigned a TEF simply because they cause measurable non-cancer effects.

- OCDD has not been shown to possess the ability to induce tumors. In fact, only 2,3,7,8-TCDD and some of the HxCDD isomers have been shown to possess the ability to induce tumors.

- OCDD has negligible capacity to bind to the Ah receptor in vitro. Given the fact that receptor-binding is considered to be a required step in the induction of dioxin-related cancer effects, the poor affinity of OCDD for this receptor is inconsistent with a non-zero TEF for OCDD. Receptor-binding contaminants were present in the OCDD studies used to derive the high-dosage TEF for OCDD. Hence, at least some of the effects observed (enzyme induction) in the OCDD feeding studies could have been caused by contaminants.

- OCDD behaves differently than 2,3,7,8-TCDD in biological tissues and specifically OCDD has a much lower oral and dermal bioavailability and, therefore, OCDD should be treated as a separate indicator chemical when assessing dioxin-related health risks.

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REVIEW AND CRITIQUE OF THE SCIENTIFIC BASIS OF THE TEF FOR OCDD

1.0 INTRODUCTION

In 1989, the USEPA revised the existing 2,3,7,8-TCDD toxic equivalent factors (TEFs) for several of the polychlorinated dibenzo-p-dioxins and dibenzo-p-furans (PCDDs and PCDFs). Included in these modifications was an increase in the TEF for octachlorodibenzo-p-dioxin (OCDD) and octachlorodibenzofuran (OCDF). The 1987 TEFs initially established by the USEPA assigned values of zero for both OCDD and OCDF, while the recently published 1989 TEFs assign a value of 0.001 to both congeners. This change will have a significant impact on the health risk estimates for sites that are contaminated primarily with these congeners. In particular, wood treatment sites, which typically contain elevated levels of OCDD in site soils could be concluded as posing significant human health risk according to the new TEF system. The purpose of this document is to review the objectives and the methodologies for establishing TEFs and to evaluate the scientific basis for the recently revised TEF values for OCDD/OCDF. In addition, the appropriate use of the new TEFs in human health risk assessment is discussed. This document is organized as follows:

SECTION 2.0 OCDD AND OCDF IN THE ENVIRONMENT. This section briefly describes how OCDD and OCDF are formed and distributed in the environment.

SECTION 3.0 PHARMACOKINETICS OF OCDD. This section provides a detailed review of the manner in which OCDD distributes in biological tissues and the associated biological responses elected in various animal studies. The results of these studies were used by the USEPA to establish the new TEFs for OCDD and OCDF. Determination of Toxic Equvialation Factor.. this section describes the methods used by USEPA to establish TEFs.

SECTION 4.0 TOXICITY OF OCDD/OCDDF. This section describes the toxic effects associated with exposure to OCDD/OCDF.

SECTION 5.0 DETERMINATION OF TOXIC EQUIVALENCY FACTORS. This section describes how the USEPA derives TEFs for the various PCDD/PCDF isomers. The basis of the new TEF for OCDD/OCDF is described and critiqued.

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SECTION 6.0 IS THE INCREASED IN THE OCDD TEF WARRANTED. This section summarizes the key elements of the previous sections and discusses how an OCDD TEF should be used in health risk assessment.

SECTION 7.0 REFERENCES

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2.0 OCDD AND OCDF IN THE ENVIRONMENT

PCDDs and PCDFs are members of a class of compounds collectively referred to as halogenated aromatic hydrocarbons. Also included in this class of compounds are polychlorinated biphenyls (PCBs), napthalenes, polybrominated biphenyls (PBBs) and azobenzenes. PCDDs, PCDFs and PCBs are highly stable, lipophilic molecules with extreme persistence in the environment (Safe, et al., 1989). PCBs were produced commercially for a number of years for use as insulation in capacitors and transformers (Hose, et al., 1986). In contrast, PCDDs and PCDFs are unwanted contaminants formed during the production of chlorinated phenols and related derivatives (Burg, 1988). These compounds are also produced during combustion processes involving organohalogen containing wastes (Safe, et al., 1989).

PCDDs and PCDFs are released into the environment via a number of pathways, including: 1) urban incinerator emissions, 2) discharge form pulp and paper mills, and 3) use of products such as pentachlorophenol (PCP) and 2,4,5-trichlorophenol contaminated with PCDDs and PCDFs (Firestone, 1977; Firestone, et al., 1972; Liberti, et al., 1980). PCDDs and PCDFs are usually found in the environment as mixtures of several different isomers. These mixtures are generally found to contain primarily the higher chlorinated congeners (hexas, heptas, and octas) (Firesonte et al., 1972). The one exception is the herbicide 2,4,5-trichlorophenol which primarily contains the 2,3,7,8-TCDD isomers (Safe et al., 1989).

PCP is used in a variety of products and is one of the most widely-used biocides in the U.S. The major use of PCP in the U. S. has been as a wood preservative. Eighty percent of the U. S. consumption is for treatment of wood used for utility poles. The toxicity of PCP was investigated by a number of research groups in the 1970's, however it is not clear whether the toxic affects observed in those experiments were the result of PCP or rather the result of impurities in the PCP (Glickman et al., 1977; Borthwich and Schimmal, 1978). In general, commercially available PCP is

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88.4% pure and is known to contain a variety of impurities, including tetrachlorophenol, chlorinated phen-oxyphenols, PCDDs, PCDFs, and polychlorinated diphenyl ethers (Firestone et al., 1972).

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3.0 PHARMACOKINETICS OF OCDD

3.1 Absorption and Distribution of OCDD Following Subcutaneous or Intraperitoneal Administration

Recently, data describing the absorption and distribution of OCDD/OCDF has been published. Brunner et al. (1989) investigated the relative distribution of a mixture of PCDDs and PCDFs in the Wistar rat. The mixture of PCDDs and PCDFs was obtained via the catalytic dechlorination/hydrogenation of OCDD and OCDF. This mixture included 2,3,7,8-TCDD as well as OCDD/OCDF and selected results are summarized in Table 3-1. The data illustrated several key points regarding OCDD. First, it is apparent from the data in Table 3-1 that the route of administration dramatically affects the distribution of OCDD as well as other PCDDs/PCDFs. Secondly, OCDD and OCDF tend to accumulate in hepatic and adipose tissue in a similar manner in this strain of rat. Moreover, OCDD and OCDF are present in the mixture in significantly higher concentrations than 2,3,7,8-TCDD (30-fold) yet the levels detected in the liver and adipose tissue are only ~ 5-10 times the level of 2,3,7,8-TCDD.

Abraham et al. (1989) recently described the uptake of OCDD/OCDF and other PCDDs and PCDFs by hepatic tissue of female Wistar rat and Marmoset monkey. These results are summarized in Table 3-2. Marmoset monkeys and female Wistar rat both retained approximately 25% of the total administered dose of 2,3,7,8-TCDD in the liver. Moreover, 2,3,7,8-TCDD in liver/adipose ratio was approximately the same for both species. OCDD uptake by hepatic tissue of both species was similar (5.8 and 7.4% for rat and monkey respectively), however, the percentages were substantially less than reported for 2,3,7,8-TCDD. In contrast to 2,3,7,8-TCDD, the liver/adipose tissue concentration ratio for both species was substantially >1 suggesting that OCDD accumulates to a greater extent in hepatic tissue than it does in adipose tissue. OCDF hepatic uptake was similar in rat and monkey, though the percent accumulating in the liver of both species was slightly greater than reported for OCDD. The liver/adipose ratio of OCDF for both species is difficult to interpret

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Table 3-1

Distribution of PCDDs and PCDFs in Female Wistar Rats Following Subcutaneous (S. C.) or Intraperitoneal (I. P.) Administration

Congener	dose (ng/kg)	ng/liver		ng/g adipose	
		s.c.	i.p.	s.c.	i.p.
Dibenzo-p-dioxin					
2,3,7,8	100	0.59-0.79	0.70-0.74	0.22-0.35	0.36-0.58
1,2,3,7,8	111	1.64-2.37	1.98-2.11	0.13-0.17	0.26-0.83
1,2,3,4,7,8	133	2.37-3.07	2.51-2.78	0.08-0.09	0.40-0.75
1,3,7,8	184	nd	nd	0.01-0.03	0.08-0.25
OCDD	**3347**	**4.67-5.47**	**9.29-18.14**	**0.22-0.43**	**4.90-13.57**
Dibenzofuran					
2,3,4,7,8	74	0.82-1.30	1.17-1.20	0.02-0.03	0.10-0.32
1,2,3,4,7,8	328	4.27-6.52	4.64-5.44	0.09-0.13	0.80-1.64
1,2,3,7,8	124	0.16-0.22	0.10-0.24	0.03-0.04	0.18-0.52
1,2,3,6,7,8	328	5.56-8.05	5.74-6.58	0.12-0.15	1.01-1.70
2,3,7,8	45	0.04-0.05	0.03-0.04	0.03-0.03	0.05-0.09
1,2,4,7,8	111	nd	nd	0.01-0.02	0.05-0.45
1,2,3,6	126	nd	nd	0.01	0.02-0.05
OCDF	**2217**	**5.53-8.55**	**7.82-16.60**	**0.09-0.19**	**3.11-6.90**

*table adapted from Brunner, et al., 1989

Table 3-2

Concentration of Various PCDDs/PCDFs Following a Single S.C. Injection: Levels After Seven Days In Wistar Rat and Marmoset Monkey*

Congener	Wistar Rat % of admin. dose in whole liver†	Wistar Rat concen. ratio liver/adipose†	Marmoset Monkey % of admin. dose in whole liver†	Marmoset Monkey concen. ratio liver/adipose†
2,3,7,8-TCDD	26.5 ± 1.25	2.4 ± 0.26	24.5 ± 4.56	1.09 ± 0.76
1,2,3,7,8-PeCDD	71.1 ± 6.43	13.1 ± 0.60	44.3 ± 5.64	2.70 ± 1.07
1,2,3,4,7,8-HxCDD	80.6 ± 3.29	31.7 ± 0.78	48.4 ± 5.79	14.2 ± 7.57
OCDD	**5.8 ± 0.15**	**17.4 ± 5.61**	**7.4 ± 1.81**	**5.6 ± 4.24**
2,3,7,8-TCDF	3.77 ± 0.90	1.6 ± 0.23	21.5 ± 2.29	-
2,3,4,7,8-PeCDF	53.4 ± 7.86	42.6 ± 2.4	57.6 ± 4.79	10.1 ± 1.93
1,2,3,6,7,8-HxCDD	77.9 ± 7.77	48.8 ± 8.69	66.1 ± 4.54	14.8 ± 2.09
OCDF	**11.7 ± 1.73**	**46.2 ± 17.3**	**15.3 ± 1.32**	**19.2 ± 17.57**

*table adapted from Abraham, et al., 1989
†data not averaged in original manuscript; averaged here for comparison

008865

because of the wide range of values in the data. However, it appears as if OCDF, like OCDD accumulates in greater quantities in the liver as opposed to adipose tissue.

3.2 Absorption and Distribution of OCDD Following Oral Exposure

3.2.1 Mixtures of PCDDs and PCDFs

Van Den Berg, et al. (1987) recently investigated the absorption and distribution of dioxins from a fly ash mixture containing PCDDs and PCDFs including OCDD/OCDF. This experiment consisted of administering the fly ash mixture orally to female Wistar rats on days 10 to 17 of pregnancy and during the first 10 days of lactation. Specific congeners were then measured in tissues of the dams, fetuses and offspring. Approximately 37% of the 2,3,7,8-TCDD in the mixture was retained in the hepatic tissue of the dams. These data are comparable to the data in Table 5 utilizing the S.C. injection protocol. Lactation did not significantly affect retention of 2,3,7,8-TCDD in the hepatic tissue of the dams as 38% of the administered dose was retained in these animals.

The fly ash mixture used by Van Den Berg contained 64% more OCDD than did the mixture used by Abraham et al. (1989). However, the results were comparable to those reported by Abraham et al. (1989) as only 7% of the OCDD and OCDF in the fly ash mixture was retained in the hepatic tissue of the dams (see Table 3-2). As with 2,3,7,8-TCDD, lactation did not significantly affect the retention of OCDD and OCDF in the hepatic tissue of the dams. OCDD and OCDF congeners were also detected in the offspring, as approximately 0.7% of the administered dose of OCDD and OCDF was retained in the hepatic tissue. Lower chlorinated PCDDs and PCDFs were detected in the fetuses, however the amount retained in the whole fetus was very small (less than 0.13%). Analysis for OCDD and OCDF in the whole fetus was not possible because of mass interference. Accumulation of OCDD in adipose tissue of the dams during pregnancy or lactation was less than 2% of the total administered dose of OCDD. Similarly, 0.89 and 0.30% of

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the total administered dose of 2,3,7,8-TCDD accumulated in the adipose tissue of the dams during pregnancy and lactation respectively.

3.2.2 Oral Administration of Purified OCDD/OCDF

Norback et al. (1975) investigated the distribution of OCDD in rats using a radiolabeled analog ([35S]-thioheptachlorodibenzo-p-dioxin). Following administration of the analog for three weeks (100 µg/day for 21 days), it was observed that over 50% of the radiolabeled analog that was retained in the body was located in various organelles of the liver. Other target tissues included adipose tissue, skin, heart, kidney, serum, lungs, testes and muscle. However, if the rats were treated with the same protocol (100 µg/day for 21 days) followed by a 6 week recovery period, the analog was detected in only hepatic tissue, skin and adipose tissue. However, because the structure-dependent activity of PCDDs and PCDFs, utilization of such an analog makes interpretation of the data difficult.

Birnbaum and Couture (1988) have also recently described the absorption and distribution of OCDD in male Fischer 344 rats. This group observed that oral absorption of OCDD never exceeded 10% of any dose, with doses ranging from 500 to 5,000 µg/kg. Moreover, absorption of OCDD was reported to be non-linear between doses of 500 and 5000 µg/kg. The authors also point out that OCDD rapidly partitions out of the blood and into various target tissue including liver, adipose tissue and skin observed that repeated administration of OCDD (50 µg/kg daily for 10 days) resulted in the accumulation of OCDD in hepatic and adipose tissue and to a lesser degree in skin and blood. Moreover, in the companion paper (Couture et al., 1988), administration of 65 doses of 50 µg/kg resulted in substantial accumulation of OCDD in hepatic and adipose tissue as well as skin and blood. Couture et al. (1988) further investigated the oral absorption of OCDD using different volumes of carrier material (corn oil). The results of this experiment are described in Table 3-3. The results in Table 3-3 indicate that the greater the volume of the carrier the greater the absorption of OCDD in male Fischer 344 rats.

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Table 3-3

Effect of Dosing Volume on Hepatic Accumulation of OCDD

Dosing Volume	ng OCDD/g liver
Experiment 1 (50 µg/kg)	
1 ml/kg**	348.1 ± 17.2
5 ml/kg***	2323.0 ± 158.4
Experiment 2 (11.5 µg/kg)***	
1 ml/kg	5.2 ± 1.0
2 ml/kg	17.8 ± 5.2
3 ml/kg	32.6 ± 8.5
4 ml/kg	69.5 ± 7.1
5 ml/kg	38.3 ± 13.3

* table adapted from Couture, et al., 1988
**rats were dosed with 50 µg/kg daily for 10 days and terminated 3 days following the last exposure
***rats were administered a single dose of 11.5 µg/kg of OCDD and terminated 3 days later

Table 3-4

Distribution of OCDD and OCDF in Rats When Administered Via Oral Ingestion

Congener/ Dose (ppb)	% of total dose in whole liver		Concentration ratio liver/fat	
	13 weeks	26 weeks	13 weeks	26 weeks
OCDF (80)	3.7	2.6	62	39
OCDF (800)	5.2	4.0	56	64
OCDD (80)	3.3	2.3	21	19
OCDD (800)	3.6	3.3	40	39
2,3,7,8-TCDD (1 ppb)	7.6	3.9	2.5	2.6

*table adapted from Wermelinger, et al., 1990

Table 3-5

Elimination of OCDD From Selected Tissues From Male Rats Following an i.v. Injection of 50 µg/kg

Tissue	Pool Size (% total dose)	Slope (days^{-1})	t 1/2 (days)
OCDD (50 µg/kg)			
Liver	72.68 ± 32.55	-0.008 ± 0.002	84
Adipose	7.13 ± 0.93	-0.018 ± 0.007	38
Skin	8.95 ± 2.21	-0.253 ± 0.094	3
	0.31 ± 0.57	+ 0.016 ± 0.041	69

*table adapted from Birnbaum and Couture, 1988

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A recent report by Wermelinger et al. (1990) described results of a 9 month feeding study with OCDD and OCDF in rats. Concentrations of 80 and 800 ppb were administered via the feed chow. Since no data had existed up to this point on the absorption of OCDF (excluding studies involving mixtures of PCDDs and PCDFs) this study is of significant importance. Distribution of OCDD and OCDF was measured in two tissues namely, hepatic and adipose tissue. The percent of the total dose in the whole liver of OCDF (80 ppb) was 3.7 and 2.6% at 13 and 26 weeks respectively. The percentage of OCDF detected in hepatic tissue increased slightly with the higher dose of OCDF (800 ppb) as 5.2 and 4.0% of the total dose of OCDF was detected in the whole liver at 13 and 26 weeks respectively. However, for the both the low and high dose of OCDD (80 and 800 ppb) the percent of the total dose found in the whole liver remained approximately 3%. While the % of the total dose of both OCDD and OCDF were not substantially different, significant differences were observed in the distribution of these congeners between hepatic and adipose tissue. The data is also summarized in Table 3-3. The data in Table 3-3 indicates that both OCDD and OCDF preferentially accumulate in hepatic tissue as opposed to adipose tissue. Both OCDD and OCDF tend to accumulate in hepatic tissue more than 2,3,7,8-TCDD. This observation was also made by Abraham et al. (1989) following S. C. injections of fly ash mixtures of PCDDs and PCDFs in male rats.

3.3 Metabolism and Elimination of OCDD and OCDF

Very little kinetic data is available for OCDD and no kinetic data is available for OCDF. Birnbaum and Couture (1988) recently reported a whole-body half-life for OCDD of 3-5 months. This is contrast to 2,3,7,8-TCDD which has a whole-body half-life of 31 ± 6 days in Sprague-Dawley rat (following a 1.0 μg/kg oral dose) (Rose et al., 1976). Table 3-5 summarizes the elimination of OCDD from liver and adipose tissue as well as the skin.

Birnbaum and Couture (1988) suggest that the long half-life of OCDD in the liver could result in the accumulation of the congener over time. In

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addition the authors urge caution in interpreting the skin elimination parameters as only 5 data points were used to arrive at these values. Norback et al. (1975) utilizing rats and a radioactive analog of OCDD reported that 90% of the administered dose was recovered in the feces as unabsorbed material. This group also reported a small lipid soluble fraction in urine. Similar results were reported by Birnbaum and Couture (1988). Feces was reported to be the major route of elimination following administration of OCDD with very little OCDD (less than 0.2%) eliminated via urine. Very little data has been reported on the metabolism of OCDD. Birnbaum and Couture (1990) reported that no [14C]-labeled CO2 or other volatiles were detected in male rats exposed to [14C]-OCDD. Moreover, this group reported that TLC analysis of extracts from tissues and feces of animals administered [14C]-OCDD revealed a single spot that migrated with an OCDD standard. This group suggests that some metabolism of OCDD is occurring as radioactivity was detected in the urine of the rats. However, the authors report that due to the low specific activity of the [14C]-OCDD confirmation of a metabolite was not possible (Birnbaum and Couture, 1990).

3.4 Biochemical Effects of OCDD/OCDF

One of the most well characterized Ah receptor-mediated affects is the induction of cytochrome P450-dependent monooxygenases (Whitlock, 1987). The induction by PCDDs and PCDFs of these isozymes is routinely measured fluorometrically via aryl hydrocarbon hydroxylase (AHH) or ethoxyresorufin O-deethylase (EROD) activity. Bradlaw, et al., (1980) investigated the induction of AHH activity in H-4-II E rat hepatoma cells using a variety of PCDDs and PCDFs including OCDD. Surprisingly, OCDD was found to weakly induce AHH activity in these cells. Mason et al. (1986) utilizing H-4-II E rat hepatoma cells estimated the EC50 (the concentration of OCDD needed to induce AHH activity 50%) for OCDD to be > 1 x 10-4 M. However, several investigators have shown that OCDD administered to rats in multiple doses over an extended period of times will induce EROD activity substantially beyond control levels. For example, Birnbaum and Couture (1988) administered 50 ug/kg of OCDD daily by gavage (5 days/week for 13

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weeks) and observed a 40-fold increase in EROD activity over control groups. The levels of OCDD that accumulated in the liver were approximately 1,000 times the levels of 2,3,7,8-TCDD that are required to produce 40-fold EROD indication. This effect was recently confirmed by Wermelinger et al. (1990). This group reported that in rats administration of 800 ppb of OCDD in the feed for 9 months caused a significant increase in EROD activity. The induction of EROD activity by OCDD (800 ppb) approached 40 to 50% of that induced by 2,3,7,8-TCDD (rats fed 1 ppb of 2,3,7,8-TCDD for 9 months).

Ahlborg et al. (1989) also has investigated the biochemical affects of OCDD with somewhat different results. Sprague-Dawley rats were administered either OCDD, 2,3,7,8-TCDD or OCDD plus 2,3,7,8- TCDD by gavage and terminated 4 weeks later. The results are summarized in Table 3-6. Administration of OCDD to Sprague-Dawley rats did not cause any statistically significant induction of hepatic EROD activity, increase hepatic P450/448 content or increased UDPGT activity. However, this is not surprising as this experiment utilized a single administration protocol. However, OCDD and 2,3,7,8-TCDD treatment did cause a significant reduction in total vitamin A content of the liver. Coadministration of OCDD and 2,3,7,8-TCDD did not result in any substantial additive affects.

In summary:

- OCDD and OCDF tend to accumulate in the liver to greater degree than 2,3,7,8-TCDD.
- OCDD is poorly absorbed (less than 10% of any administered dose) from the gastrointestinal tract and absorption decreases with decreasing dosing volume.
- OCDD has a biological half-life of 3-5 months.
- The liver concentrations of OCDD that are required to produce 40-fold EROD induction are approximately 1,000 times the liver concentrations of 2,3,7,8-TCDD required to produce similar induction.

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Table 3-6

Effects of 2,3,7,8-TCDD, OCDD and 2,3,7,8-TCDD plus OCDD in Sprague-Dawley Rats

Parameter	control (corn oil)	2,3,7,8-TCDD (2.5 µg/kg)	OCDD (755 µg/kg)	2,3,7,8-TCDD + OCDD
Vitamin A (µg)				
Liver	1269	509[a]	837[a]	550[a]
Kidney	4.5	12.0	3.4	16.3[a]
Hepatic P450/448**	0.42	0.59	0.32	0.67[a]
EROD†	0.08	1.00[a]	0.09	1.03[a]
UDPGT††	0.06	0.34	0.25	0.34[a]

*table adapted from Ahlborg, et al., 1989

**nmol/mg M. P.

†nmol/mg M.P./min; EROD = ethoxyresorufin O-deethylase

††nmol/mg M. P. • min; UDPGT = UDP-glucoronosyl transferase

[a]statistically significant difference ($p < 0.05$) from untreated control calculated by Tukey's HSD test.

008872

4.0 TOXICITY OF OCDD AND OCDF

Non-cancer Effects

Schwetz, et al. (1973) investigated the toxicity of OCDD in both rats and mice. Oral administration (single dose) of OCDD to 5 female Sprague-Dawley rats (1 g/kg) or to 4 male Swiss Webster mice (4 g/kg) did not cause any deaths. Moreover, no overt signs of toxicity were observed in these animals. However, King, et al. (1973) reported that all male and female Osborne-Mendel rats (35 per group) fed chow containing 1 or 0.5% of OCDD died within 32 weeks. Interestingly, this group observed that male B6C3F1 mice were much more susceptible to OCDD induced toxicity as male mice fed a diet containing 1 or 0.5% OCDD all died within 10 weeks. Similar experiments in female B6C3F1 resulted in 5 survivors (from a group of 50) in the 1% group and 45 survivors (from a group of 50) in the 0.5% treatment group. However, these results should be interpreted with caution as King et al. (1973) reported that approximately 0.1% of the OCDD was contaminated with hexachlorodibenzo-p-dioxin (substitution pattern not specified). Moreover, contamination of 0.1% by hexachlorodibenzo-p-dioxin (HxCDD) was reported to result in an intake of 150 ug/kg-day of HxCDD. King et al. (1973) states that new mice-feeding experiments utilizing OCDD (0.5% in the feed) free of hexachlorodibenzo-p-dioxin contamination have also resulted in a substantial number of deaths.

Schwetz et al. (1973) reported that OCDD tested negative for production of chloracne in the rabbit bioassay. Moreover, OCDD administered at 100 and 500 mg/kg-day (2.5 ml/kg by gavage) to female Sprague-Dawley rats on from days 6 through 15 of gestation did not result in any increase in fetal resorptions, changes in fetal body measurements or fetal anomalies. However, a significant increase in subcutaneous edema was detected in the fetuses at the 500 mg/kg-day dose. Chicks fed 0.1 and 0.5% OCDD in the diet (0.1% = 100 mg/kg) showed no signs of toxicity following 20 to 21 days of treatment as determined by the chick edema biossay. It should be noted that while this study is somewhat dated, the information provided by Schwetz et al. (1973) concerning other congeners, (in which a great

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deal is known) such as 2,3,7,8-TCDD, appears to be correct. For example, this report correctly identified guinea pigs as the most susceptible species to 2,3,7,8-TCDD toxicity. Moreover, limited studies with the relatively non-toxic 2,7-dichlorodibenzo-p-dioxin also suggest that while the study is dated the results can be considered valid. Ahlborg et al. (1989) recently reported that administration of 755 ug/kg of OCDD to Sprague-Dawley rats did not cause significant body weight loss. Moreover, neither liver or thymus weights were significantly affected by OCDD administration.

Couture et al. (1988) also investigated the toxic affects of OCDD in male rats. Treatment related cytoplasmic fatty vacuolization of the liver (centrilobular and midzonal regions) were observed upon administration of 50 ug/kg of OCDD 5 days/week for 13 weeks (65 administrations total). It was noted that hepatic damage did not begin to occur until 40 doses of OCDD had been administered. Moreover, the cytoplasmic fatty vacuolization increased in severity as the number of doses increased from 40 to 65. Couture et al. (1988) also reported that OCDD did have some slight affects of blood chemistry including decreases in MCH, MCV, HGB and HCT. The authors suggest that these results may imply "mild, nonregenerative anemia resulting from a chronic, noninfectious inflammatory process. This is most likely a secondary affect of the other alterations in the liver."

Kerkvliet et al. (1985) reported that mice orally administered 100 or 500 ug/kg of OCDD (single dose) showed no decrease in the humoral immune response as evidenced by the splenic IgM response to sheep erythrocytes (SRBC). The splenic IgM response to sheep erthrocytes (SRBC) is considered one of the most sensitive assays in detecting toxicity elicited by PCDDs and PCDFs.

008874

Cancer Effects

The only carcinogenic data available pertaining to OCDD were reported by King et al. (1973). The affect of OCDD as a complete carcinogen and as a promoter were investigated using mouse skin tumor formation as an endpoint. Swiss-Webster mice (30 female and 30 male) were "painted" 3 times weekly with OCDD (0.2 ml of a 0.2 mg/ml solution for 60 weeks) to test for complete carcinogenic activity. To test for promotional activity, the mice were first treated with 50 µg of dimethylbenzanthracene (DMBA) prior to exposure to OCDD for 59 weeks. No papillomas or carcinomas were detected in either experimental protocol. However, in the complete carcinogenesis study, 1 subcutaneous tumor was observed in each of the sexes. In the promotional study, 3 subcutaneous tumors were observed in the male mice and 1 was observed in female mice. The total dose of OCDD administered to the rats was approximately 7080 µg (0.2 mg/ml x 0.2 ml/treatment x 3 treatments/week x 59 weeks = 7080 µg). Hebert et al. (1990) recently investigated the tumor promoting activity of 2,3,7,8-TCDD, 2,3,4,7,8-pentachlorodibenzofuan (2,3,4,7,8-PeCDF) and 1,2,3,4,7,8-hexachlorodibenzofuran (1,2,3,4,7,8-HxCDF) and demonstrated that all three congeners were potent promoters of squamous cell carcinomas. Interestingly, the dose of OCDD used by King et al. (1973) was approximately 177 times greater than that of 1,2,3,4,7,8-HxCDF (largest dose was 1 ug/treatment x 2 treatments/week x 20 weeks = 40 µg). Based on the 1989 TEFs, OCDD would be predicted to be 100 times less potent than 1,2,3,4,7,8-HxCDF. However, OCDD was shown not to be a promoter of skin carcinogenesis in the Swiss-Webster mouse model using a dose of OCDD that should have produced skin tumors at a comparable rate to that of 1,2,3,4,7,8-HxCDF and 2,3,7,8-TCDD (King et al., 1973). It should be noted that Hebert et al. (1990) used the hairless mice model for the promotion studies while King et al. (1973) used the Swiss-Webster mouse model and the discrepancies noted above may be simply a result of species differences. Clearly, more research is warranted to investigate the tumor promoting activities of OCDD and OCDF.

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In summary:

- OCDD does not appear to be acutely toxic and possesses weak teratogenic, immunotoxic, and carcinogenic properties.
- In the only cancer study performed to date with OCDD, OCDD failed to demonstrate the carcinogenic potential expected from a chemical with a TEF of 0.001.

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5.0 DETERMINATION OF TOXIC EQUIVALENCY FACTORS

Evaluating the human health risks associated with exposure to PCDDs and PCDFs in the environment is difficult due to a lack of data describing the toxicity of each individual congener. In the environment and in PCP, PCDDs and PCDFs are found in complex mixtures containing any number of the 210 possible congeners, which further complicates the human health risk assessment process.

5.1 Backgroumd

Because of the difficulties in assessing the human health risk from exposure to mixtures of PCDDs and PCDFs, the USEPA devised a system to assign potency values for the many different congeners possible in these mixtures. This system ranks the potency of individual congeners relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) (Barnes, et al., 1989; Safe, 1987; Safe, et al., 1989). 2,3,7,8-TCDD was chosen as the standard as numerous studies have identified this congener as the most toxic member of the PCDDs and PCDFs (Poland and Knutson, 1982). These potency values are termed toxic equivalent factors (TEF) and are derived from in vitro and in vivo data. The type of data used to derive TEFs includes but is not limited to the following; 1) induction of cytochrome P450-dependent monooxygenases in both human and rodent cell culture as well as in animals, 2) immunotoxicity in mice, 3) body weight loss in a variety of animal models, 4) reproductive toxicity in mice and 5) receptor-binding studies. These are non-cancer biological responses that are produced upon exposure to 2,3,7,8-TCDD. Other dioxin isomers can also cause these effects to a lesser degree. TEFs are assigned to specific dioxin isomers based on their potency (relative to 2,3,7,8-TCDD) at inducing these affects. As described in Section 3.0, the OCDD concentrations in liver required to achieve maximal enzyme induction are 1,000 times that of 2,3,7,8-TCDD. Hence, OCDD is considered to be 1/1,000 as potent as 2,3,7,8-TCDD and has been assigned a TEF of 0.001.

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It is important to emphasize that while the TEFs determined from the assays above are used in human carcinogenesis risk assessments, the endpoints measured in these assays are not in themselves related to carcinogenicity. Specifically, although 2,3,7,8-TCDD is known to produce tumors in chronically exposed animals, almost none of the other dioxin and furan isomers have been assayed for tumorigenic potential in a long-term feeding study. Nevertheless, many of the non-2,3,7,8-TCDD isomers are assumed to have some tumorigenic potential due to their ability to produce non-cancer biochemical responses (such as liver enzyme induction) similar to 2,3,7,8-TCDD. Given the expense and practical limitations associated with performing long-term cancer bioassays for each individual isomer, using non-cancer end points as indicators of carcinogenic potential is considered the most reasonable approach for assessing health risks for dioxin mixtures. The above-mentioned non-cancer responses are believed to be linked to the same events that are responsible for tumor developments. Hence, relative potencies based on non-cancer effects are considered to be accurate measures of relative carcinogenic potencies.

To obtain a 2,3,7,8-TCDD toxic equivalency value for a given congener in a mixture, the TEF value established by EPA is multiplied by the concentration of the individual congener in a mixture. For example, the current TEF for octachlorodibenzo-p-dioxin (OCDD) is 0.001. Therefore, 20,000 ppb of OCDD is treated as 20 ppb of 2,3,7,8-TCDD for risk assessment and remediation purposes (20,000 x 0.001 = 20). This process is repeated for each individual congener or each separate class of congeners and the values (2,3,7,8-TCDD toxic equivalents) are then summed to arrive at an estimate of the toxic potency of the mixture. In 1989, EPA revised the 1987 TEFs. Table 5-1 illustrates the distinct differences between the 1987 and 1989 TEF values. These include: 1) values of zero were assigned to all non-2,3,7,8- substituted congeners, 2) different TEFs for 1,2,3,7,8- and 2,3,4,7,8- PeCDFs, 3) increasing the TEF value for 2,3,7,8- substituted hexa- and hepta- congeners, and 4) increasing the OCDD/OCDF TEF from zero to 0.001. Approximately 75% of the "dioxin" detected in the environment is the highly chlorinated OCDD (NRCC, 1981).

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Table 5-1
Comparison of the 1987 EPA-TEF's and the Revised 1989 I-TEF's[1] for PCDDs and PCDF's*

Congener	EPA-TEF's (1987)	I-TEF's (1989)
Mono-, Di-, and TriCDD	0	0
2,3,7,8-TCDD	1	1
other TCDDs	.01	0
2,3,7,8-PeCDDs	0.5	0.5
other PeCDDs	0.005	0
2,3,7,8-HxCDDs	0.04	0.1
other HxCDDs	0.004	0
2,3,7,8-HpCDDs	0.001	0.01
other HpCDDs	0.0001	0
OCDD	**0**	**0.001**
Mono-, Di-, and TriCDF	0	0
2,3,7,8-TCDF	0.1	0.1
other TCDFs	.001	0
1,2,3,7,8-PeCDF	0.1	0.05
2,3,4,7,8-PeCDF	0.1	0.5
other PeCDDs	0.00	0
2,3,7,8-HxCDFs	0.01	0.1
other HxCDFs	0.0001	0
2,3,7,8-HpCDFs	0.001	0.01
other HpCDFs	0.00001	0
OCDF	**0**	**0.001**

[1]international -TEFs
*table adapted from Barnes, et al., 1989

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As mentioned previously, OCDD is the primary isomer in many dioxin sources, and increasing the TEF values for OCDD could significantly impact the health risk estimates for many sites throughout the U.S.

5.2 Proposed Mechanism of Action of PCDDs and PCDFs and its Relationship to TEFs

5.2.1 Receptor Binding and Structure-Activity Relationships

The toxic and biologic responses elicited by PCDDs and PCDFs are proposed to occur via a receptor-mediated mechanism. The receptor protein that mediates the action of PCDDs and PCDFs was identified in 1976 and was termed the aryl hydrocarbon (Ah) receptor (Poland, et al., 1976). To be a true receptor-mediated event certain criteria must be met including (Clark, et al., 1988):

- specific binding by prospective ligands
- high affinity binding of the ligand with the receptor
- saturable binding by specific ligands
- correlation with a biological event
- tissue and species specificity

The Ah receptor has been shown to meet all of the above criteria (Poland and Knutson, 1982; Goldstein and Safe, 1989). The Ah receptor has been associated with a number of biological responses elicited by PCDDs and PCDFs including: teratogenicity, induction of cytochrome P450-dependent monooxygenases, body weight loss, thymic atrophy, regulation of endocrine systems including estrogen and progesterone receptor levels and immunotoxicity (reviewed in Goldstein and Safe, 1989). The mechanism by which PCDDs and PCDFs exert their affects is similar to the mechanism proposed for steroid hormones. PCDDs and PCDFs are lipophilic molecules that passively diffuse across the cellular membrane and into the cytosolic compartment of the target cell (Poland and Knutson, 1982). Once in the

008880

cytosol, the congener or ligand binds to the Ah receptor which results in both the transformation of the receptor into a DNA binding protein and the translocation of the ligand-Ah receptor complex into the nucleus of the cell (Harris, et al., 1989a; Harris, et al., 1989b; Okey, et al., 1979; Okey, et al., 1980). The "nuclear" ligand-Ah receptor complex then interacts with specific DNA sequences termed dioxin responsive elements (DREs) which results in the initiation of transcription. This mechanism is well characterized for the induction of cytochrome P450-dependent monooxygenases. Specific DREs have been identified in the 5-flanking region of the cytochrome P450 gene and binding of the ligand-Ah receptor complex to DREs has been demonstrated (Israel and Whitlock, 1984; Israel and Whitlock, 1983; Whitlock, 1986; Whitlock, 1987; Whitlock, 1988; Whitlock, 1989; Whitlock and Galeazzi, 1984).

Numerous investigations have established that the effects elicited by PCDD or PCDF administration are structure-dependent. Specifically, the ability of each individual dioxin is isomer to elicit the above effects depends on the placement of the chlorine atoms on the dioxin structure. It has been clearly established that the PCDDs and PCDFs chlorinated in the lateral positions (2,3,7,8- positions) are the most potent with respect to producing biological responses (see Table 5-1). 2,3,7,8-TCDD has been identified as the most active congener of the PCDDs and PCDFs (Goldstein and Safe, 1989; Mason, et al., 1[illegible] Mason, et al., 1985). Table 5-2 illustrates that as chlorination increases beyond the 4 lateral substituents for PCDDs, the potency of the congeners decreases. Moreover, removal of lateral substituents also causes a significant reduction in potency. Consistent with the criteria stated above for a receptor-mediated response, the 2,3,7,8- substitution pattern correlates with high affinity binding for the Ah receptor (Table 5-3). Moreover, removal of lateral or addition of non-lateral substituents dramatically affects binding to the Ah receptor. The structure-activity and structure-binding rules for PCDFs vary slightly from the rules established for PCDDs. Because of the structure of the dibenzofuran molecule, the addition of an additional substituent in the C-4 position results in a

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Table 5-2
A Summary of the In Vivo Biologic and Toxic Effects of Several PCDD and PCDF Congeners in Male Wistar Rat

Congener	In Vivo ED_{50} (μmol/kg) *		
	Inhibition of Body Weight Gain	Thymic Atrophy	AHH Induction
2,3,7,8-TCDD	0.05	0.09	0.004
2,3,7,8-TBDD	0.068	0.034	0.00036
1,2,3,7,8-PeCDD	0.62	0.17	0.031
1,2,3,4,7,8-HxCDD	1.63	1.07	0.03
1,3,7,8-TCDD	132	100	31.2
1,2,4,7,8-PeCDD	34.0	11.0	2.82
2,3,7,8-TCDF	3.2	3.60	0.652
2,3,4,7,8-PeCDF	1.04	0.21	0.037
1,2,3,7,8-PeCDF	2.64	1.76	1.47
1,3,4,7,8-PeCDF	26.1	0.70	3.49
1,2,3,6-TCDF	> 250	>250	>160

*table adapted from Goldstein and Safe (1989)

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spatial conformation more similar to that of 2,3,7,8-TCDD (reviewed in Goldstein and Safe, 1989).

5.2.2 Correlation of TEFs with Ah Receptor Binding

Table 5-3 illustrates several points of interest with regard to the TEF system. In general, congeners that compete with [3H]-2,3,7,8-TCDD for binding to the Ah receptor have been assigned TEF values that reflect this characteristic. These compounds are typically substituted in the lateral 2,3,7,8- positions (e.g., 2,3,4,7,8-PeCDF). In contrast, congeners that have a reduced binding affinity for the Ah receptor such as 1,2,4,7,8-PeCDF are assigned reduced TEF values and these congeners generally are not completely substituted in lateral positions.

However, Table 5-3 contains two instances where the competitive EC50 values for the Ah receptor are inconsistent with the corresponding TEF value. These congeners are 2,3,7,-trichlorodibenzo-p-dioxin (2,3,7-TriCDD) and OCDD. As described above, moderate to high competitive binding for the Ah receptor by a given congener generally correlates with a non-zero TEF. However, for at least one congener, namely 2,3,7-TriCDD, this is not the case. Recent studies utilizing [3H]-2,3,7-TriCDD suggest that following i.p. administration to C57BL/6 mice, the radioligand fails to accumulate in hepatic tissue to any appreciable degree. Moreover, the small amount of [3H]-2,3,7-TriCDD that is retained in hepatic tissue does not form nuclear [3H]-2,3,7-TriCDD-Ah receptor complexes (Harris, et al., 1990). Consequently, although 2,3,7-TriCDD is able to bind to the Ah receptor, its complete lack of activity in vivo is reflected in its TEF value of zero.

The case for OCDD is substantially different. The EC50 for OCDD binding to the Ah receptor is > 10,000 nM, which indicates that OCDD has little to no affinity for the Ah receptor in vitro. However, OCDD has been assigned a TEF value of 0.001, based on its ability to cause maximal liver enzyme induction at a liver concentration 1,000 times that of 2,3,7,8-TCDD. There are several possible explanations for the inconsistency

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Table 5-3

Structure-Binding Activities for Selected Dibenzo-p-dioxins and Dibenzofurans in Rat Hepatic Cytosol

Congener	Lateral Chlorines	Non-lateral Chlorines	EC_{50} Receptor Binding values (nM)**	1989 I-TEF Values
Dibenzo-p-dioxin				
2,3,7,8	4	0	10	1
1,2,3,7,8	4	1	79	0.5
2,3,6,7	3	1	160	0
2,3,6	2	1	220	0
1,2,3,4,7,8	4	2	280	0.1
1,3,7,8	3	1	790	0
2,3,7	3	0	71	0
2,8	2	0	3200	0
1,2,3,4,5,6,7,8 (OCDD)	4	4	>10,000	0.001
Dibenzofuran				
2,3,4,7,8	4	1	15	0.5
1,2,3,4,7,8	4	2	230	0.1
1,2,3,7,8	4	1	74.5	0.05
1,2,3,6,7,8	4	2	270	0.1
2,3,7,8	4	0	41	0.1
1,2,4,7,8	3	2	1300	0
1,2,3,6	2	2	354	0

* data adapted from Mason, et al., 1986; Mason, et al., 1985

** amount needed to displace 50 % of [^{3}H]-2,3,7,8-TCDD (10 nM) in a competitive binding assay.

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between the lack of OCDD receptor-binding activity and the assigned TEF. Couture et al. (1988) recently suggested the lack of receptor-binding by OCDD in vitro could be a function of its high degree of chlorination and subsequent insolubility. Conversely, it may be possible that the liver enzyme induction observed in the OCDD feeding studies were: 1) independent of receptor-binding and/or 2) caused by a receptor binding contaminant present in the OCDD. If the enzyme induction was truly independent of receptor binding, the carcinogenic potential of OCDD would need to be seriously re-evaluated. Receptor binding is critical for translocation of the chemical into the nucleus. If the chemical cannot bind to the Ah receptor in vivo, then it has no carcinogenic potential (as discussed above for 2,3,7-TriCDD). Probably the best method for resolving this in consistency between lack of receptor binding in vitro and enzyme induction in vivo would be to measure ^{14}C-OCDD binding to the Ah receptor in vivo (as has been done for 2,3,7-TricCDD). If no binding occurred, then OCDD would be given a TEF of zero. It is also possible that a contaminant caused the enzyme induction in the OCDD feeding studies. Indeed, Birnbaum and Couture (1988) mention that several dioxin isomers were detected in their OCDD, but they felt that the levels of these trace contaminants were insufficient to cause significant enzyme induction. This issue may warrant further study. In summary:

- The cancer and non-cancer effects elicited by 2,3,7,8-TCDD and the other PCDDs/PCDFs are thought to be dependent on binding to the Ah receptor.

- Only the 2,3,7,8-TCDD isomer and a mixture of some Hx CDDs have been shown to produce tumors in animals.

- TEFs for the other isomers are assigned based on their relative potency (compared to 2,3,7,8-TCDD) with respect to non-cancer effects such as receptor binding and enzyme induction.

- OCDD has not been shown to bind to the Ah receptor in vitro, yet has been assigned a TEF of 0.001 because the liver concentrations of OCDD required to produce maximal enzyme induction in vivo are approximately 1,000 times the required 2,3,7,8-TCDD concentrations.

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- The inconsistency between lack of receptor binding in vitro and the enzyme induction in vivo suggests that 1) the enzyme induction observed in vivo with OCDD may not have been receptor-initiated and/or 2) contaminants with receptor binding capacity were present in the OCDD used in the feeding studies. In either case the carcinogenic potential of OCDD would need to be seriously questioned.

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6.0 IS THE INCREASE IN THE OCDD TEF VALUE WARRANTED?

It is clear from the studies noted above that OCDD and OCDF act in a "dioxin" like manner under specific dosing regimens (Couture, et al., 1988; King, et al., 1973; Schwetz, et al., 1973; Wermelinger et al., 1990). These specific conditions such as chronic exposure to small concentrations are not unlike the types of exposures both workers and residents generally experience. Moreover, these congeners tend to accumulate in target tissue such as the liver and like other PCDDs and PCDFs are not readily metabolized (Abraham, et al., 1989; Birnbaum and Couture, 1988; Couture, et al., 1988, Neubert, et al., 1990; Schwetz, et al., 1973; Wermelinger, et al., 1990). King et al. (1973) demonstrated OCDD was negative as both a promoter and initiator of skin carcinogenesis in the Swiss-Webster mouse model. In addition, the dose used by King et al. (1973) was approximately 177 times greater than that used by Hebert et al. (1990) for 1,2,3,4,7,8-HxCDF in hairless mice and should have produced tumors at a comparable rate. Clearly based on the data of Hebert et al (1990) and King et al. (1973) there is a discrepancy in the carcinogenic potency of OCDD and the current TEF.

The TEF values of 0.001 assigned to both OCDD and OCDF are warranted if certain assumptions are correct, including:

1. Enzyme and receptor binding assays used to determine TEFs for PCDDs and PCDFs are accurate predictors of carcinogenic potency. There is evidence that this may not be the case.

2. The mechanism of action of PCDDs and PCDFs is the same in humans and animals.

The selection of a TEF for OCDD of 0.001 reflects the rule established by numerous structure-activity studies namely, increased chlorination in non-lateral positions deceases the potency of the congener. The doses used by Couture et al. (1988) and Wermelinger et al. (1990) to obtain near maximal EROD induction were approximately 500-1000 fold greater than the amount of 2,3,7,8-TCDD needed to induce EROD activity to the same extent.

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Consequently, the USEPA established a TEF of 0.001 for OCDD. However, it is important to emphasize that there are no data which indicate that OCDD is a carcinogen in animals or humans. It is recommended that additional studies be conducted to investigate the carcinogenic potential of OCDD and OCDF as there is evidence in the literature that suggests these congeners are not promoters of the carcinogenic process.

In addition, it is important to note that OCDD differs from 2,3,7,8-TCDD with respect to absorption and distribution in biological tissues. Specifically, it is known that the oral bioavailability of pure OCDD is at least an order of magnitude lower than that of 2,3,7,8-TCDD (Birnbaum and Couture, 1988). Dermal bioavailability is likely to be lower by a corresponding margin. As described in the USEPA's "1989 Update to procedures for estimating dioxin risks":

> "The TEFs for the highly chlorinated congeners ignore the issue of relative bioavailability of the CDD/CDF congeners, which have not been thoroughly investigated. Lower relative bioavailability of the hepta- and octa- forms compared to the tetra- forms would generally reduce the concern for TEQ estimates for samples such as those which are dominated by the hepta- and octa- forms. Research in this area is needed to resolve this point.'
>
> 'In samples taken from biological organisms exposed to PCP-contaminated soils in Region IX, the TEQs were within a factor of two of each other, when calculated by the I-TEF/89 method or the EPA-TEF/87 method. Although the data are limited, they appear to suggest that the differences in TEQs observed in the PCP-contaminated soil samples are not observed in tissues or organisms exposed to this soil."

Accordingly, it would be inaccurate to treat OCDD concentration as simply a fraction of 2,3,7,8-TCDD in risk assessment. OCDD should be treated as a specific chemical of concern, with its own bioavailability factors, when assessing health risks at dioxin contaminated sites.

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In summary:

- OCDD does not appear to bind to the Ah receptor in vitro. Receptor binding is considered to be a critical step in the carcinogenic process of dioxin.
- A TEF of 0.001 for OCDD/OCDF was recently established by the USEPA because OCDD is approximately 1/1000 as potent as 2,3,7,8-TCDD in causing maximal enzyme induction in rat liver.
- Receptor-binding contaminants were known to be present in the OCDD used in the high-dosage animal feeding studies.
- The TEF is based strictly on non-cancer effects, there are no data indicating that OCDD is a carcinogen.
- OCDD should be treated as a specific indicator chemical at dioxin sites.

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APPENDIX C

APPENDIX C

Dioxin Clean-up Goals for Arkwood, Inc. Site

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DIOXIN CLEANUP GOALS FOR ARKWOOD

During past discussions regarding potential dioxin cleanup levels for soils at the Arkwood site, it has been suggested that "a remedial goal of 20 ppb dioxin is appropriate for Arkwood since this level has been used at other industrial Superfund sites throughout the U.S." It is important to emphasize that simply adopting a cleanup level from another site is inappropriate and completely ignores recent USEPA guidance for developing soil remediation goals at Superfund sites. In 1989, the USEPA issued a guidance document that has been prepared expressly for the purpose of establishing soil cleanup levels at individual Superfund sites. The document, entitled "Guidance for Establishing Target Cleanup Levels for Soils at Hazardous Waste Sites" (1989) provides detailed guidance for deriving health-based, site-specific cleanup levels for chemicals in soil. The main objectives of the guidance document are: 1) to ensure that site-specific factors are considered during development of remediation criteria, and 2) to avoid the use of arbitrary, non-specific cleanup criteria.

As described in the guidance document, the remediation goal established for a particular site is usually not directly applicable to another. A scientifically defensible, health-protective remediation goal for any site requires a detail analysis of several site-specific factors, including: distance to the nearest human receptor, meteorological conditions, the nature of the dioxin present at the site, soil type, depth of contamination, etc. Adoption of a cleanup level established at a different site provides no guarantee that the remediation criteria will be health-protective.

The above mentioned cleanup level of 20 ppb was initially developed primarily by Dr. Renate Kimbrough of the Centers for Disease Control (CDC) for a dioxin site in Missouri. This cleanup level was not a health-based level, as it was not derived through a careful, thorough evaluation of

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potential exposures and health risks. Accordingly, it was not the intent of the CDC that this level be routinely applied to other dioxin sites without consideration of site-specific conditions. As described in the Record of Decision (ROD) for the Syntex Agribusiness site in Verona, Missouri:

> "Kimbrough recommends that risk management decisions by EPA should be based upon a consideration of the specific circumstances and exposure opportunity at each contaminated site. Kimbrough notes that in certain nonresidential areas, higher dioxin levels may present an acceptable degree of health risk."

In summary, use of a remediation goal from another site without consideration of site-specific factors is inappropriate and inconsistent with USEPA guidance. In order to ensure consistency with the current practice of risk assessment and accepted methodologies for establishing remediation criteria for soil, it would be appropriate to derive a cleanup level for dioxin at Arkwood in a manner consistent with the 1989 EPA guidance document, as opposed to simply adopting a cleanup level from another site.

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APPENDIX D

Alternatives to Incineration for Soils Containing Greater than 20 ppb Dioxin

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ALTERNATIVES TO INCINERATION FOR SOILS CONTAINING >20 PPB DIOXIN

It has been suggested that, based on historical precedence, incineration is the only acceptable remedial alternative for soils containing >20 ppb dioxin. However, a review of several Records of Decisions for dioxin sites throughout the U.S. indicates that several alternatives to incineration have been used for soils containing greater than 20 ppb dioxin.

Some of these are summarized below:

- Diamond-Alkali: 80 Lister Avenue - New Jersey (1988). Soils containing >20 ppb dioxin will be capped on-site.
- Selma Pressure Treating Co. - California (1988). Soils containing >20 ppb will be bonded with cement and covered on-site with a RCRA cap.
- Broderick Wood Products - Adams County, Colorado (1988). Soils containing >20 ppb dioxin will be stored on-site in a single-lined and covered waste pile.

In general, if the site is industrial and is relatively inaccessible, dioxin levels of >20 ppb are acceptable, as long as measures are taken to prevent migration from the site and/or direct human contact. As described in a memo (5/8/87) from Vernon Houk, Assistant Surgeon General for the Centers of Disease Control (CDC) to Barry Johnson, Associate Administrator for the Agency for Toxic Substances and Disease Registry:

> "For industrial areas, it is acceptable to leave surface concentrations of greater than 20 ppb under the paved surface. This would require continued monitoring for integrity of the paved surface where the average dioxin levels exceeding 20 ppb are left under the pavement."

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Accordingly, it would be inaccurate to assume that industrial soils containing >20 ppb dioxin must be incinerated to protect public health. There is well-established precedent in which such soils at Superfund sites have been remediated with alternative methods.

Given the fact that 1) dioxin-related health risks at Arkwood are estimated to be on the order of 10^{-5}, and 2) the average dioxin concentrations at Arkwood range from 10 - 70 ppb, it is apparent that soil levels of >20 ppb dioxin would be health protective for Arkwood.

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APPENDIX E

APPENDIX E

Calculation of Preliminary Health-Based Soil Cleanup Levels for Polynuclear Aromatic Hydrocarbons (PAHs) at the Arkwood Site in Omaha, Arkansas

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PRELIMINARY ESTIMATES OF HEALTH-BASED SOIL CLEANUP LEVELS FOR POLYNUCLEAR AROMATIC HYDROCARBONS (PAHs) AT THE ARKWOOD SITE IN OMAHA, ARKANSAS

INTRODUCTION

This document provides preliminary health-based cleanup levels for PAHs in soils at the Arkwood, Inc. site in Omaha, Arkansas. These cleanup levels are 'health-based', in that these levels of PAH in soil would not present a significant health risk to the exposed populations described in the 1989 Endangerment Assessment (EA). The derivation of health-based soil cleanup levels contained herein follows the methodology prescribed by the "Guidance for Establishing Target Cleanup Levels For Soils at Hazardous Waste Sites" (USEPA, 1988). This guidance divides the current task into four steps:

1) Definition of Exposure Scenario
2) Exposure Assessment
3) Dose-response Assessment for Chemicals of Concern
4) Dose Allocation and Calculation of Target Cleanup Levels

The exposure assumptions described in this document are identical to those used in the 1990 Evaluation of the Arkwood Risk Assessment, with the exception of the dermal and oral bioavailabilities, which were derived herein specifically for the PAHs. The EPA has accepted risk levels of one in one hundred thousand (10^{-5}) and greater at sites where the size of the receptor population would probably result in very infrequent exposures. For the purposes of this assessment, health-based soil cleanup levels are calculated for two risk levels: 10^{-6} and 10^{-5}.

1.0 DEFINITION OF EXPOSURE SCENARIOS

As described in the 1989 EA, the most probable future land use conditions involves unrestricted access to the Main Site as well as the Railroad Ditch (Exposure Scenario II). The 1989 EA considered child and adult exposure at the Main Site and exposure of railroad personnel, children, and adults at the Railroad Ditch. These populations are also considered in this document.

2.0 EXPOSURE ASSESSMENT

2.1 Fate and Transport Assumption

PAHs have been shown to be susceptible to various degradative processes in soil. However, for the purpose of deriving health-based soil cleanup levels, it will be assumed that PAH levels in soil remain constant.

2.2 Pathways and Routes of Exposures

Exposure pathways are the means through which an individual may come into contact with a chemical in the environment (e.g., drinking contaminated water from a well). Routes of exposure describe the means through which the chemical gains entry to the body via a particular pathway (e.g., dermal absorption of a soil-bound chemical while gardening). This section describes all of the potential pathways and routes of human exposure to the PAHs at the Arkwood site. The quantitative assessment of exposure, combined with a knowledge of the PAH concentration present at the site, provides the basis for estimating daily PAH uptake and any associated health risks. The 1985 EA identified several pathways of exposure to PAHs at the Arkwood site. These pathways included dermal and oral absorption of soil-bound PAHs. PAHs were not detected in the groundwater at the site. The exposure parameters developed in the following sections are summarized in Table 2-1.

2.2.1 Exposure Via Ingestion

Soil Ingestion

Exposure to contaminants at the Arkwood site may occur through ingestion of contaminated soil either during recreational or occupational activities. Ingestion of soil generally is confined to children between the ages of 1.5 to 6 years of age and this is a result of the tendency of children at this age to mouth and chew foreign objects. Moreover, several factors can influence this behavior including nutritional and economic status as well as race (Charney, et al., 1980). A variety of estimates

Table 2.1 - Lifetime Average Daily Dose (LADD) Equations
Carcinogenic Polynuclear Aromatic Hydrocarbons

EXPOSURE SCENARIO: MAIN SITE
Exposure Pathway: Oral

RECEPTOR POPULATION (group)	SOIL INGESTION RATE (mg / exposure event)	EXPOSURE FREQUENCY (exposure events/year)	CONVERSION FACTOR (kg/mg)	BIOAVAILABILITY (unitless)	EXPOSURE DURATION (years)	TIME CONVERSION FACTOR (years/day)	1/BODY WEIGHT (1/kg)
CHILD	5.00	6.00	1.00E-06	0.40	6.00	2.74E-03	3.45E-02
ADULT	5.00	12.00	1.00E-06	0.40	58.00	2.74E-03	1.43E-02
LIFETIME							

Exposure Pathway: Dermal

RECEPTOR POPULATION (group)	SKIN SURFACE AREA (cm2/event)	AMOUNT OF SOIL ADHERING TO SKIN (mg/cm2)	EXPOSURE FREQUENCY (exposure events/year)	CONVERSION FACTOR (kg/mg)	BIOAVAILABILITY (unitless)	EXPOSURE DURATION (years)	TIME CONVERSION FACTOR (years/day)	1/BODY WEIGHT (1/kg)
CHILD	625.00	0.50	6.00	1.00E-06	0.006	6.00	2.74E-03	3.45E-02
ADULT	840.00	0.50	12.00	1.00E-06	0.006	58.00	2.74E-03	1.43E-02
LIFETIME								

EXPOSURE SCENARIO: RAILROAD AREA
Exposure Pathway: Oral

RECEPTOR POPULATION (group)	SOIL INGESTION RATE (mg / exposure event)	EXPOSURE FREQUENCY (exposure events/year)	CONVERSION FACTOR (kg/mg)	BIOAVAILABILITY (unitless)	EXPOSURE DURATION (years)	TIME CONVERSION FACTOR (years/day)	1/BODY WEIGHT (1/kg)
CHILD	5.00	6.00	1.00E-06	0.40	6.00	2.74E-03	3.45E-02
ADULT	5.00	12.00	1.00E-06	0.40	58.00	2.74E-03	1.43E-02
LIFETIME							
RR PERS.	5.00	6.00	1.00E-06	0.40	30.00	2.74E-03	1.43E-02

Exposure Pathway: Dermal

RECEPTOR POPULATION (group)	SKIN SURFACE AREA (cm2/event)	AMOUNT OF SOIL ADHERING TO SKIN (mg/cm2)	EXPOSURE FREQUENCY (exposure events/year)	CONVERSION FACTOR (kg/mg)	BIOAVAILABILITY (unitless)	EXPOSURE DURATION (years)	TIME CONVERSION FACTOR (years/day)	1/BODY WEIGHT (1/kg)
CHILD	625.00	0.50	6.00	1.00E-06	0.006	6.00	2.74E-03	3.45E-02
ADULT	840.00	0.50	12.00	1.00E-06	0.006	58.00	2.74E-03	1.43E-02
LIFETIME								
RR PERS.	840.00	0.50	6.00	1.00E-06	0.006	30.00	2.74E-03	1.43E-02

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Table 2.1 - Lifetime Average Daily Dose (LADD) Equations (CONT.)
Carcinogenic Polynuclear Aromatic Hydrocarbons

EXPOSURE SCENARIO: MAIN SITE

Exposure Pathway: Oral

RECEPTOR POPULATION (group)	1/AVERAGING TIME (1/years)	SOIL INTAKE FACTOR (kg soil/kg bw-day)	HYPOTH[illegible]ON SOIL CONCE[illegible]ON (mg/kg)	LADD (mg/kg/day)	30 YR. RESID. LADD (mg/kg/day)
CHILD	1.43E-02	9.73E-11	1.00E+02	9.73E-09	4.17E-09
ADULT	1.43E-02	7.80E-10	1.00E+02	7.80E-08	3.34E-08
LIFETIME		8.77E-10	1.00E+02	8.77E-08	3.76E-08

Exposure Pathway: Dermal

RECEPTOR POPULATION (group)	1/AVERAGING TIME (1/years)	SOIL INTAKE FACTOR (kg soil/kg bw-day)	HYPOTHETICAL SOIL CONCENTRATION (mg/kg)	LADD (mg/kg/day)	30 YR. RESID. LADD (mg/kg/day)
CHILD	1.43E-02	8.36E-11	1.00E+02	8.36E-09	3.58E-09
ADULT	1.43E-02	9.01E-10	1.00E+02	9.01E-08	3.86E-08
LIFETIME			1.00E+02	9.84E-08	4.22E-08

EXPOSURE SCENARIO: RAILROAD AREA

Exposure Pathway: Oral

RECEPTOR POPULATION (group)	1/AVERAGING TIME (1/years)	SOIL INTAKE FACTOR (kg soil/kg bw-day)	HYPOTHETICAL SOIL CONCENTRATION (mg/kg)	LADD (mg/kg/day)	30 YR. RESID. LADD (mg/kg/day)
CHILD	1.43E-02	9.73E-11	1.00E+02	9.73E-09	4.17E-09
ADULT	1.43E-02	7.80E-10	1.00E+02	7.80E-08	3.34E-08
LIFETIME		8.77E-10	1.00E+02	8.77E-08	3.76E-08
RR PERS.	1.43E-02	2.02E-10	1.00E+02	2.02E-08	8.64E-09

Exposure Pathway: Dermal

RECEPTOR POPULATION (group)	1/AVERAGING TIME (1/years)	SOIL INTAKE FACTOR (kg soil/kg bw-day)	HYPOTHETICAL SOIL CONCENTRATION (mg/kg)	LADD (mg/kg/day)	30 YR. RESID. LADD (mg/kg/day)
CHILD	1.43E-02	9.12E-11	1.00E+02	9.12E-09	3.91E-09
ADULT	1.43E-02	9.83E-10	1.00E+02	9.83E-08	4.21E-08
LIFETIME			1.00E+02	1.07E-07	4.60E-08
RR PERS.	1.43E-02	2.54E-10	1.00E+02	2.54E-08	1.09E-08

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have been proposed for the amount of soil ingested by young children. Originally, the CDC proposed as much as 10 g/day of soil was ingested by children between the ages of 1 and 3.5 while children 3.5 and 5 years of age were assumed to ingest 1g/day of soil. However, Paustenbach et al. (1986) calculated that if these assumptions by CDC were correct, then 80% of the entire lifetime dose of a non-volatile, hydrophobic chemical present in soil would occur during the first 5 years of life (Paustenbach, et al., 1986). Other groups have attempted to estimate the soil intake by children including: NRC, 40 mg/day; Day et al. (1975), 100 mg/day; Bryce-Smith (1974), 33 mg/day; Hawley (1985), 100 mg/day.

Recently, Calabrese et al. (1989) conducted a rigorous study to determine the amount of soil that was typically ingested by children. This group utilized the measurement of tracer elements in the feces of 64 healthy children between the ages of 1 and 4 years. This study was more definitive than prior investigations because it analyzed the diet of the children, assayed for the presence of tracers in the diapers, assayed house dust and surrounding soil, and corrected for the pharmacokinetics of the tracer materials. Eight tracer elements were measured in this study though three elements namely, Si, Al and Y gave the best percentage of recovery (close to 100%) and the lowest standard deviation. Based on the results of two of the three most reliable tracers, the amount of soil ingestion by children (2-4 years of age) was found to average approximately 25 mg/day.

Adults do not generally ingest soil and thus their intake of potentially contaminated soil would be quite low (Vermeer and Frate, 1979). Moreover, even taking into account poor hygiene and eating soil-contaminated food, the 100 mg/day figure suggested by CDC for use in estimating soil intake by adolescents and adults seems high. A figure of 5 mg/day is reasonable if, as some have suggested, adults ingestion 10% of the amount of dirt eaten by children. Therefore, the following soil ingestion rates will be used to derive health-based soil cleanup levels.

008908

Age Group (years)	Ingestion Rate (mg/day)
0-6	25
6-12	5
12-70	5

The ingestion rate of 25 mg/day is derived from Calabrese's (1989) tracer study, and the 5 mg/day value is based on the assumption that adults and adolescents ingest soil at a rate of 10% that of children.

2.2.2 Exposure via Dermal Absorption

Soil Adherence

A parameter that can effect the dermal absorption of PAHs is the amount of soil adhering to human skin. Lepow et al. (1975) using adhesive tape to sample a defined area of skin suggested that the amount of soil adhering to human skin was approximately 0.5 mg/cm^2 (Lepow, et al., 1975). Furthermore, the California Department of Health Services Toxic Substances Control Division estimated that approximately 0.9 mg/cm^2 of soil was adhering to the hands of children (average age was 11) (Services, 1986). In addition, several studies have been published which describe the effect of soil particle size and organic content of soil on adherence of soil to human skin. For example, Que Hee et al. (1985) using a variety of soils of different particle size suggested that on average, about 0.2 mg/cm^2 of soil was adhering to the hands of small adults (Que Hee, et al., 1985). Driver et al. (1989) surveyed various soils of different organic content for their ability to adhere to human hands (adult male). It was determined that the average amount of soil adhering to human hands was 0.6 mg/cm^2 (unsieved). Thus, the available literature suggests that the amount of soil adhering to the hands of humans resides somewhere between 0.2 and 0.9 mg/cm^2 (Driver, et al., 1989). Therefore, for the purposes of establishing health-based soil cleanup levels, a value of 0.5 mg/cm^2 seems a reasonable estimate for the amount of soil adhering to the hands of humans.

2.3 Bioavailability of Polynuclear Aromatic Hydrocarbons (PAHs)

Environmental contaminants are able to cross biological barriers with varying degrees of efficiency. The bioavailability (% of applied dose that is systemically absorbed) of an environmental contaminant is governed primarily by 1) the physico-chemical properties of the contaminant, 2) the environmental matrix in which it is present, and 3) the nature of the biological membrane. Chemicals in soil are usually absorbed to a lesser degree than the chemicals in pure form. Bioavailability factors for ingested contaminants in water or soil typically fall in the range of 20-30%, while dermal absorption factors for chemicals in water or soil are often less than 10%.

Bioavailability factors are typically estimated from studies in which chemicals uptake has been measured in animals exposed to the chemical of concern (in pure form and/or bound to soil). In the absence of any exposure data involving the chemical of concern, it is usually appropriate to examine the results of exposure studies performed with similar chemicals. For example, many risk assessments involving PCB exposure assume that the oral bioavailability of PCBs in soil is 40%. This assumption is based on a number of ingestion studies performed with soil-bound dioxin, a chemical which possesses many physico-chemical characteristics similar to the PCBs and is known to be approximately 40% absorbed from soil upon ingestion.

The estimate of bioavailability of soil-bound PAHs is an important parameter in the risk assessment process. While several investigators have reported bioavailability figures for some PAHs administered in pure form, little information is available on the bioavailability of soil-bound PAHs.

The sections below describe the oral and dermal bioavailability factors used to derive the health-based cleanup levels for PAHs (as a group).

2.3.1 Oral Bioavailability

Oral bioavailability is a measure of the degree to which a chemical will be systemically absorbed following ingestion. Some chemicals are absorbed almost completely (100% bioavailability) when ingested in pure form. Other chemicals may pass through the body largely unabsorbed. In general, as the lipophilicity of a chemical increases, its absorption across the gastrointestinal tract increases.

Oral bioavailability of soil-bound chemicals is also dependent on the rate at which chemicals dissociate from the soil matrix in the gut. Soil-bound chemicals are usually absorbed to a lesser degree than a chemical in pure form. The reduced absorption is a result of a hydrophobic attraction between the chemical and the soil matrix. The greater the degree of affinity between chemical and soil, the less likely it is that soil-bound chemical will be absorbed upon ingestion.

The degree to which soil-bound PAHs are absorbed upon ingestion has not been researched. However the oral bioavailability of soil-bound PAHs can be estimated based on the results of research involving compounds with similar physical properties, primarily soil binding coefficients (Koc). The soil binding coefficient is the measure of the tendency for organic chemicals to be absorbed to soil. It is expressed as follows:

$$Koc = \frac{\text{grams of chemical absorbed to soil/g organic chemical}}{\text{grams of chemical dissolved in solution/ml of solution}}$$

A high Koc value (10^5-10^7 ml/g) indicates that the chemical has an affinity for soil, and that the chemical will likely remain bound to soil even in the acidic environment of the gastrointestinal tract.

The chemical tetrachlorodibenzo-p-dioxin (TCDD) is a compound for which much research has been performed. Its soil binding coefficient (Koc) is 3,300,000 ml/g (USEPA, 1986b), which is in the range of Kocs for PAHs (10^4 - 10^7 ml/g). The Koc value for BaP is 5,500,000 /g (USEPA, 1986b). Site-specific oral bioavailability factors for dioxin in soil and fly-ash have been determined for several sites (Shu et al., 1988; Umbreit et al., 1985; Lucier et al., 1986). Those values have been used in several risk

assessments, including the Centers of Disease Control (CDC) health risk assessment for Times Beach, Missouri where dioxin in soil was assumed to be 50% bioavailable (Kimbrough et al., 1984). The feeding studies of Poiger and Schlatter (1980) and McConnell et al. (1984) provided the basis for this estimate. The results of those studies have also been used to estimate bioavailabilities for similar chemicals in numerous risk assessments (Paustenbach, 1986).

An oral bioavailability factor of 50% was used for PAHs in the 1989 EA. Similarly, for the purposes of this risk assessment, 40% oral bioavailability is assumed for PAHs in a soil matrix. This assumption is supported by the fact that, like dioxin: PAHs are readily absorbed in the gut when administered in pure form or in solution (Drill et al., 1981), and PAHs are similar to dioxin in that they generally have very low vapor pressures, very low water solubilities, and most importantly, a very high affinity for soil as noted by their high Koc values.

2.3.2 Dermal Bioavailability

For a chemical to be absorbed through the percutaneous route, it must pass through the stratum corneum, the epidermis, the dermis and into systemic circulation. In contrast, compounds absorbed by the lungs or gastrointestinal tract may pass through only two cells to be systemically absorbed (Klaasen et al., 1986). It is generally accepted that diffusion throughout the stratum corneum is the rate limiting factor for dermal absorption (Scheuplein and Blank, 1971; Schaefer et al., 1983; McLaughlin, 1984). It should be kept in mind that absorption is a time-dependent phenomenon since chemicals move across the stratum corneum by passive diffusion. For the purposes of this assessment, it will be conservatively assumed that contaminated soil will be in contact with skin for 12 hours before the soil is washed from the skin.

The vehicle in which the chemical is delivered to the skin can have a decided impact on the efficiency of absorption. The ability of a compound to penetrate the skin is dependent on two consecutive physical events. The compound must first diffuse or desorb from the vehicle to the skin

surface, and then must penetrate the skin (Ostrenga et al., 1971). Absorption of contaminants from a medium such as soil and dust if inhibited by physical chemical bonding to the soil matrix and because only a small portion of the contaminant is in direct contact with the skin (Hawley, 1985).

Yang et al. (1989) measured percutaneous absorption of Benzo(a)pyrene (BaP) absorbed to soil contaminated with petroleum crude oil. The percent recovery was measured at 24 hour intervals after the application of a "monolayer" of 1% petroleum crude-fortified soil on the skin of female rats. The authors note that 1) soil applied in excess of a monolayer (~9 $\mu g/cm^2$) did not increase the amount of BaP absorbed, and 2) the absorption of BaP showed little change over the applied concentration range between 0.1 and 1000 ppm. The authors reported that approximately 1.1% of the dose appeared in urine and faces 24 hours after dosing. Since it is unlikely that a significant portion of the absorbed BaP dose could have sequestered into tissues or body fat within this short time period, it is reasonable to assume that the percentage of dose measured in the urine and feces is representative of the total absorbed dose. For the purposes of this assessment, it will be assumed that 0.55% of the PAH concentration in soil that adheres to skin is absorbed during a 12-hour period [1.1% x (12 hrs/24 hrs)].

Applying the results of rat studies is a conservative approach. Several reports have suggested that rat skin may be at least 10-fold more permeable than human skin (Bartek, et al., 1972; Bartek and La Budde, 1975). Moreover, it has been reported that the difference in permeability between rat and human skin was greater for lipid-soluble compounds than for water-soluble compounds Comparison of the permeability of several compounds including the lipophilic haloprogin and water-soluble acetylcysteine gave interesting results. The rat:human dermal penetration ratio for the lipophilic haloprogin was 9:1. In contrast, the very water soluble acetylcysteine rat:human dermal penetration ratio was 1.4:1 (Bartek, et al., 1972; Bartek and La Budde, 1975). These results suggest that absorption by human skin of lipophilic chemicals is approximately 10-fold less than that of rat skin.

The 1989 EA assumed a dermal bioavailability of 1% for PAHs in soil, based on the Kimbrough estimate for dioxin. For the purposes of this assessment, a dermal bioavailability of 0.55% is assumed.

2.4 Dose Calculations

2.4.1 General Intake Equation

In this review, the total lifetime average daily dose (LADD) is used to quantitatively estimate potential cancer risks. The total LADD is the sum of the LADDs calculated for each age group. Oral and dermal LADDs for each age group are calculated according to the following general equation:

$$I = \frac{C \times CR \times EFD}{BW \times AT}$$

Where:

I - intake; the amount of chemical (mg/kg body weight-day) at the exchange boundary (e.g., skin or gastrointestinal tract)

Chemical-related variable

C - chemical concentration; the average concentration contacted over the exposure period (e.g., mg/kg soil)

Variables that describe the exposed population

CR - contact rate; the amount of contaminated medium contacted per unit time or event (e.g., kg soil/day)

EFD - exposure frequency and duration; describes how long and how often exposure occurs. Often calculated using two terms (EF and ED):

EF - exposure frequency (days/year)

ED - exposure duration (years)

BW - body weight; the average body weight over the exposure period (kg)

008914

Assessment-determined variable

AT - averaging time; period over which exposure is averaged (e.g., years)

This general equation is consistent with the general dose equation presented in the USEPA's Human Health Evaluation Manual and is consistent with the equation described in the 1989 EA.

2.4.2 Specific Intake Equations

The general intake equation is modified for soil ingestion and dermal absorption as follows:

$$\text{Ingestion Intake (mg/kg-day)} = \frac{CS \times IR \times CF \times FI \times ABS \times EF \times ED}{BW \times AT}$$

Where:

CS - Chemical Concentration in Soil (mg/kg)

IR - Ingestion Rate (mg soil/day)

CF - Conversion Factor (10^{-6} kg/mg)

FI - Fraction Ingested from Contaminated Source (unitless)

ABS - Absorption Factor (unitless)

EF - Exposure Frequency (days/years)

ED - Exposure Duration (years)

BW - Body Weight (kg)

AT - Averaging Time (period over which exposure is averaged-days)

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$$\text{Absorbed Dermal Dose (mg/kg-day)} = \frac{CS \times CF \times SA \times AF \times ABS \times EF \times ED}{BW \times AT}$$

Where:

CS - Chemical Concentration in Soil (mg/kg)

CF - Conversion Factor (10^{-6} kg/mg)

SA - Skin Surface Area Available for Contact ($cm^2$3/event)

AF - Soil to Skin Adherence Factor (mg/cm^2)

ABS - Absorption Factor (unitless)

EF - Exposure Frequency (events/year)

ED - Exposure Duration (years)

BW - Body Weight (kg)

AT - Averaging Time (period over which exposure is averaged)

Both equations consider bioavailability of the chemical in the absorption factor. Derivation of oral and dermal bioavailability values is discussed in Section 2.3. Exposure assumptions used in estimating chemical intake are listed in Table 2-1.

3.0 DOSE-RESPONSE ASSESSMENT FOR CARCINOGENIC PAHs

Certain PAHs have been shown to produce cancer in laboratory animals, although there is no evidence of carcinogenicity in humans. Indicator chemicals identified at the Arkwood site include the carcinogenic PAHs benzo(a)pyrene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene and chrysene.

For the vast majority of PAHs found at hazardous waste sites, there are inadequate data to assess carcinogenicity, and consequently the USEPA has only propagated a slope factor for benzo(a)pyrene [B(a)P] (11.53 mg/kg/day^{-1}). However, recent scientific thought indicates that assessing the carcinogenic risk of total PAHs as a group using this slope factor would certainly lead to an overestimation in estimated risk. For example, the

TABLE 2_2

EXPOSURE ASSUMPTIONS[1]

EXPOSURE FACTOR		REFERENCE CITED FOR REFINED ASSUMPTIONS
SOIL INGESTION RATE		
6-12 years	5 mg/day	Calabrese et al., 1989
12-70 years	5 mg/day	
SOIL ADHERENCE FACTOR	0.5 mg/cm^2	Driver et al., 1989
SKIN SURFACE AREA		
6-12 years	625 cm^2	U.S. EPA, 1989b
12-70 years	870 cm^2	Snyder, 1975
DERMAL BIOAVAILABILITY	.55%	Yang et al., 1989
ORAL BIOAVAILABILITY	40%	Shu et al., 1988
LIFETIME EXPOSURE DURATION		
all receptor populations	30 years	U.S. EPA, 1989b

[1] all other exposure assumptions are identical to those used in the 1989 Endangerment Assessment

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carcinogenic strength of benzo(a)anthracene is found to be about 1/2,000 of that of B(a)P. Therefore the approach used herein, which assumes that all PAHs are equivalent to BaP in potency, is extremely health-conservative.

Using the equations presented in Section 2.0, a risk-specific lifetime average daily dose (or uptake) can be calculated as follows:

$$\text{Risk-Specific Lifetime Daily Dose (LADD)} = \frac{\text{Acceptable Risk Level}}{\text{Slope Factor}}$$

For BaP, the risk-specific dose associated with a 1×10^{-5} risk equals:

$$\text{Risk-Specific LADD (mg/kg-day)} = \frac{1 \times 10^{-5}}{11.53\ (\text{mg/kg-day})^{-1}}$$

$$= 8.67 \times 10^{-7}\ \text{mg/kg-day}$$

4.0 CALCULATION OF HEALTH-BASED SOIL CLEANUP LEVELS

The dose equations presented in Section 2.0 follow the general form:

$$LADD = U * CsL$$

Where:

- LADD = Lifetime Average Daily Dose
- U = Uptake (the product of all exposure parameters)
- CsL = Contaminant Soil Concentration

To calculate the health-based soil cleanup levels, the LADD is substituted by a risk-specific dose (RsD) and the equation is solved for CsL:

$$CsL = RsD / U \qquad \text{(Eq. 4-1)}$$

Where:

- CsL = Contaminant Soil Concentration
- RsD = Risk-specific Daily Dose
- U = Uptake (the product of all exposure parameters)

The Risk-specific Dose (RsD) is calculated by dividing the acceptable increased cancer risk level (10^{-6} or 10^{-5}) by the Cancer Potency Factor (CPF). As previously discussed, the CPF for benzo(a)pyrene of 11.53 $(mg/kg/day)^{-1}$ is used in these calculations. As described below, the fractional RsD associated with each exposure pathway is used to derive the cleanup levels.

4.1 Relative Contribution of Different Pathways

The lifetime average daily dose (LADD) equations were calculated for each pathway using a hypothetical soil PAH concentration of 100 mg/kg. Dividing these numbers into the total LADD for all pathways, the relative contribution of each pathway to the total dose is determined (See Appendix A). As can be seen in Table 4-1, the contribution of both the oral and dermal pathways to overall dose is about fifty percent.

4.2 Selection of the Applicable Ceanup Levels

The derivation of health-based soil cleanup levels for carcinogenic PAHs is presented in Table 4-2. Cleanup levels are established for each fractional RsD and the lowest value is chosen as the cleanup level for the site. These cleanup levels (as presented in Table 4-2) indicate that a soil concentration of approximately 50 ppm carcinogenic PAHs would be protective of all potentially exposed populations associated with the most likely future land use scenario. It is important to note that these are very conservative cleanup levels as they assume that all of the PAHs at the Arkwood site are as carcinogenic as benzo(a)pyrene, even though this is known not to be the case.

5.0 SUMMARY

The main conclusions of this preliminary assessment may be summarized as follows:

* The exposure assumptions used in this document are taken from the primary scientific literature.

Table 4-1 Dose Fractionation

RECEPTOR POPULATION pathway	FRACTIONAL RsDs Railroad (mg/kg/day)	(%)	Main Site (mg/kg/day)	(%)
Visitors				
oral	3.76E-08	45%	3.76E-08	47%
dermal	4.59E-08	55%	4.22E-08	53%
Railroad personnel				
oral	8.66E-09	44%	NA	NA
dermal	1.09E-08	56%	NA	NA

LADD - Lifetime Average Daily Dose (estimated from exposure)
NA - Not Applicable

TABLE 4-2 : DOSE FRACTIONATION AND CALCULATION OF HEALTH-BASED SOIL CLEANUP LEVELS

Risk Level of 1E-06 :

SCENARIO receptor Pathway	LADD (mg/kg/day)	total LADD (mg/kg/day)	CONTRIBUTION (fraction)	RISK LEVEL (unitless)	CPF 1/(mg/kg/day)	RSD (mg/kg/day)	eq.RSD (mg/kg/day)	UPTAKE (kg/kg/day)	Csl (mg/kg/day)
Main Site									
oral	3.76E-08	7.98E-08	0.47	1.00E-06	1.15E+01	8.67E-08	4.09E-08	8.77E-10	46.60
dermal	4.22E-08	7.98E-08	0.53	1.00E-06	1.15E+01	8.67E-08	4.59E-08	9.85E-10	46.56
Railroad									
adult									
oral	3.76E-08	8.35E-08	0.45	1.00E-06	1.15E+01	8.67E-08	3.91E-08	8.77E-10	44.53
dermal	4.59E-08	8.35E-08	0.55	1.00E-06	1.15E+01	8.67E-08	4.77E-08	1.07E-09	44.56
rr pers.									
oral	8.66E-09	1.96E-08	0.44	1.00E-06	1.15E+01	8.67E-08	3.84E-08	2.02E-10	190.09
dermal	1.09E-08	1.96E-08	0.56	1.00E-06	1.15E+01	8.67E-08	4.82E-08	2.54E-10	189.89

Risk Level of 1E-05 :

SCENARIO receptor Pathway	LADD (mg/kg/day)	total LADD (mg/kg/day)	CONTRIBUTION (fraction)	RISK LEVEL (unitless)	CPF 1/(mg/kg/day)	RSD (mg/kg/day)	eq.RSD (mg/kg/day)	UPTAKE (kg/kg/day)	Csl (mg/kg/day)
Main Site									
oral	3.76E-08	7.98E-08	0.47	1.00E-05	1.15E+01	8.67E-07	4.09E-07	8.77E-10	465.97
dermal	4.22E-08	7.98E-08	0.53	1.00E-05	1.15E+01	8.67E-07	4.59E-07	9.85E-10	465.63
Railroad									
adult									
oral	3.76E-08	8.35E-08	0.45	1.00E-05	1.15E+01	8.67E-07	3.91E-07	8.77E-10	445.32
dermal	4.59E-08	8.35E-08	0.55	1.00E-05	1.15E+01	8.67E-07	4.77E-07	1.07E-09	445.57
rr pers.									
oral	8.66E-09	1.96E-08	0.44	1.00E-05	1.15E+01	8.67E-07	3.84E-07	2.02E-10	1900.94
dermal	1.09E-08	1.96E-08	0.56	1.00E-05	1.15E+01	8.67E-07	4.82E-07	2.54E-10	1898.92

LADD - Lifetime Average Daily Dose
CONTRIBUTION - Relative contribution to total LADD
CPF - Cancer Potency Factor
RsD - Risk-specific Dose
UPTAKE - Soil Intake factor (See Table 2.1)
Csl - Contaminant Level in Soil (CLEANUP LEVEL)

* Under the most probable future use conditions at Arkwood, approximately 50 ppm of carcinogenic PAHs in Arkwood soils would be associated with a 10^{-6} health risk. This assumes that all PAHs are as carcinogenic as benzo(a)pyrene, the most carcinogenic PAH known.

* Since many of the PAHs at Arkwood are less carcinogenic than benzo(a)pyrene, cleanup levels of much greater than 50 ppm (total carcinogenic PAHs) would still be health-protective.

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APPENDIX F

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APPENDIX F

Alternative I - Sieve and Wash/In Situ Vitrification

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The following information is provided to compare soil washing/ISV to Alternative H by using the same format as that of the FS Report.

6.2.10 Alternative I - Sieve and Wash/In Situ Vitrification

Description

In Alternative I, the site is fenced and existing structures are removed. Affected soils are excavated and treated by sieving-and-washing. The sand/fines slurry portion is pumped to the washwater treatment process described in Alternative E and dewatered. Also as in Alternative E, the coarse soils are separated into fractions for testing and filtrate is recycled to the sieve-and-wash process. A trench is excavated and backfilled with the dewatered sand/fines fraction, filter cake from the washwater processing system and sludge from the railroad ditch and the sinkhole. Contents of the trench are then processed by in situ vitrification (ISV). Alternative I provides destruction by pyrolysis of constituents of concern in the sludge and in the sand/fines fraction of the soil. Off-gases generated during the process are captured beneath a hood and delivered to an on-site treatment system. The sand/fines soil and sludge fraction contains approximately 30% of the soil mass and 80% of the PCP mass in the soils. The resultant inert monolith is left in place and backfilled with a clean fill/topsoil cap to accommodate subsidence resulting from a reduction in volume of approximately 30% to 40% with the ISV process. The sinkhole fluids and equipment decontamination water are treated by carbon adsorption. The carbon is then disposed of by incorporation in one of the melts. The remainder of the site is covered with a topsoil cap.

To construct the ISV system, a trench is excavated in an area above the water table and away from any areas of concern related to karst geology features. The trench is lined with plastic. Then sand/fines filter cake and sludge are transported to and backfilled into the trench. Electrodes are placed in the affected material at a spacing sufficient to treat an area approximately 25 ft square in plan. A hood is erected over the area to capture volatile emissions and the ISV process is commenced. Electric power is applied to melt the soil mass by heating it to temperatures of 1,600°C to 2,000°C. Three hours after the initial melt area is completed, the subsidence zone is backfilled and the hood is moved to cover the next treatment area while the first is cooling. The process is repeated until all material deposited in the trench has been solidified into an inert, obsidian-like mass which requires no further treatment and has no potential for migration.

Previous studies by GEOSAFE, INC. on various ISV treated wastes shown that chlorinated organics decompose to their basic elements

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of carbon, hydrogen and chlorine. This process will achieve results which, under appropriate application conditions, exceed incineration performance standards and meet EPA's EP-Toxicity and TCLP criteria.

Operation of the sieve-and-wash system is as described in Alternative F. The sand/fines will be removed and backfilled in the trench with the filter cake before starting the ISV process. Consideration will be given to the possibility of treating the deeper locations of affected material, such as at the railroad siding, in place without excavation. The ISV process is designed to operate on a continuous basis until all material in the trench has been treated.

Prior to full scale operation, treatability tests are required to optimize ISV system performance.

Design Basis

See Section 6.1.3 for basis of fencing, decontamination and removal of existing structures, sieve-and-wash, ground water monitoring and topsoil cap.

Volume excavated	20,400 c.y.
Area of topsoil cap	650,000 s.f.

In situ vitrification design parameters

Constituents of concern	PCP, Dioxin, PAH
Heat value of affected material	Substantial in sludge which will require dilution with other soil.
Required cleanup levels	300 ppm PCP, 20 ppb dioxin as 2,3,7,8, TCDD equivalents
Soil type/composition	Clay with chert nodules (approximately 60% of volume); chemistry not yet evaluated.
Soil moisture content	Assume 25%
Volume to be treated	7,000 to 8,000 c.y.

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Dry density	Assume 1.3 ton/c.y.
Depth of processing	Assume 15 ft, depending upon site specific conditions
Tonnage to be treated	10,400 tons, based upon assumed density
Quantity of material treated per setting	
Volume	Approximately 400 c.y.
Tonnage	Approximately 520 tons
Expected melt dimensions	15 ft x 25 ft x 25 ft
Construction Period	6 months to 1 year
Operating Period	6 months to 1 year

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7 - EVALUATION OF ALTERNATIVES FOR SLUDGES AND AFFECTED SOILS

(See FS Report, Volume I, May 23, 1990)

7.2 Overall Protection of Human Health and the Environment

Alternative I (Sieve-and-Wash/In Situ Vitrification) is rated "++" since it provides the greatest reduction in residual risk. The reasons are that ISV provides destruction and removal of organic compounds, vitrification of inorganic compounds, and total emissions control to permanently eliminate the pathway of exposure.

7.3 Compliance with ARARs

Alternative I (Sieve-and-Wash/In Situ Vitrification) is rated "." because it complies with ARARs.

7.4 Long Term Effectiveness and Permanence

Alternative I (Sieve-and-Wash/In Situ Vitrification) is rated "++" for this criterion because with the ISV process, it is possible to achieve maximum possible destruction. Additionally, the vitrified mass is inert, analogous to an obsidian petrology.

7.5 Reduction of Toxicity, Mobility, or Volume

Alternative I (Sieve-and-Wash/In Situ Vitrification) is rated "++" for the following reasons:

a. ISV results in complete destruction of contaminants at operating temperatures,
b. A volume reduction of 20% to 40% is achieved with ISV,
c. Treatability tests will indicate no leaching from the vitrified mass, and
d. The process is irreversible.

7.6 Short-Term Effectiveness

Alternative I (Sieve-and-Wash/In Situ Vitrification) is rated "+". Compared to on-site incineration, the amount of soil handling and resultant exposure, is somewhat less. Moreover, there is less overall exposure with ISV due to the shorter project duration and because there is total emissions control with ISV.

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7.7 Implementability

Alternative I (Sieve-and-Wash/In Situ Vitrification) is rated " - ". The comments related to the sieve-and-wash system for Alternative E are appropriate for this selection. Additionally, the system requires specialized equipment and skilled personnel for operation. However, compared to the construction of a complex, on-site incineration system, the equipment required for ISV is available, mobile and easily installed. Trained personnel are also available. At this time, the Arkwood, Inc. site has voltage capability to allow an electrical load sufficient for the ISV process. However, the power company would have to replace approximately two miles of conductor. Project duration is reduced because of less time required for permitting and no trial burn. Additionally, there is no ash to handle, no additional soils remedial actions needed, and ISV will not interfere with ground water remedial actions.

7.8 Cost

Alternative I (Sieve-and-Wash/In Situ Vitrification) is rated "-" for this criterion. Costs for in situ vitrification are reduced when proceeded by sieve-and-wash. Compared to incineration, costs are reduced because:

a. The equipment is trailer mounted, for the most part, reducing costs for mobilization and demobilization,
b. Permitting, verification sampling and analysis costs are reduced
c. Overhead costs are reduced because of the shorter project duration.

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TABLE 6-1

COMPARISON OF ESTIMATED COST AND TIME TO COMPLETION
FOR REMEDIAL ALTERNATIVES FOR SLUDGES AND AFFECTED SOILS

Arkwood Inc Site
Omaha, Arkansas

Alternative	Cost [a] (millions)	Time to Completion (years)
A NO Action	$0.29	---
B Site Monitoring and Restricted Access	$0.4	0.3
C Incinerate Sludges	$2.1	0.5
C1 Incinerate Sludges / Topsoil Cap over Entire Site	$3	0.5
D Incinerate Sludges / Consolidate and Cap-in-Place Affected Soils	$4	1.0
E Incinerate Sludges / Consolidate, Sieve-and-wash and Cap-in-Place Affected Soils	$6.6	2.0
F Incinerate Sludges / Sieve-and-Wash Biologically Treat Fines and Cap-in-Place Affected Soils	$14	4.0
G Incinerate Sludges / Landfill Affected Soils On-Site	$5.5	2.0
H Incinerate Sludges and Affected Soils On-Site	$18	3.0
I Sieve-and-wash/In-Situ Vitrification	$11.8	2.0

NOTES

[a] Costs are net present value based on the following

- Assumes an interest (discount) rate of 5% (net of inflation) and a 30-year post-closure care period
- The sum of capital and operating costs and the net present value of the post-closure care costs
- Costs are mid-1989

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TABLE 6-11

CAPITAL COST SENSITIVITY ANALYSIS

Arkwood Inc Site
Ohana, Arkansas

Alternative [b]	Net Present Value of Alternatives Costs (Millions of Dollars) [a]				
	50%	30%	0%	-20%	-30%
A NO ACTION	0 29	0 29	0 29	0 29	0 29
B Site Monitoring and Restricted Access	0 44	0 43	0 41	0 39	0 39
C Incinerate Sludges	3 0	2 7	2 1	1 8	1 6
D Incinerate Sludges Topsoil Cap Over Entire Site	4 4	3 9	3 1	2 5	2 3
E Incinerate Sludges / Consolidate and Cap-in-Place Affected Soils	6 0	5 2	4 1	3 4	3 0
F Incinerate Sludges / Consolidate Sieve-and-Wash and Cap-in-Place Affected Soils	9 0	8 4	6 6	5 3	4 7
G Incinerate Sludges / Sieve-and-Wash Biologically Treat Fines and Cap-in-Place Affected Soils	20	18	14	11	10
H Incinerate Sludges / Landfill Affected Soils On-Site	5 0	7 0	5 5	4 5	4 0
I Incinerate Sludges and Affected Soils On-Site	27	23	18	14	13
Sieve-and Wash/In-Situ Vitrification	17 6	15 3	11 8	9 6	8 4

NOTES

a) Using 5% discount rate and 30 year post-closure care and monitoring period

b) Percent change in capital costs from those given in tables 6-2 through 6-10

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TABLE 6-12

POST-CLOSURE CARE COST SENSITIVITY ANALYSIS

Arkwood Inc Site
Omaha Arkansas

Alternative	(b)	Net Present Value of Alternatives Costs (Millions of Dollars) [a]				
		50%	30%	0%	-20%	-30%
A No Action		0 44	0 38	0 29	0 23	0 20
B Site monitoring and Restricted Access		0 58	0 51	0 41	0 34	0 31
C Incinerate Sludges		2 3	2 2	2 1	2 1	2 0
D Incinerate Sludges / Topsoil Cap over Entire Site		3 3	3 2	3 1	3 0	3 0
E Incinerate Sludges / Consolidate and Cap-in-Place Affected Soils		4 3	4 2	4 1	4 0	4 0
F Incinerate Sludges / Consolidate Sieve-and-Wash and Cap-in-Place Affected Soils		6 7	6 7	6 6	6 5	6 4
G Incinerate Sludges / Sieve-and-Wash Biological Treatment and Cap-in-Place Affected Soils		14	14	14	14	13
H Incinerate Sludges / Landfill Affected Soils On-Site		5 7	5 6	5 5	5 4	5 4
I Incinerate Sludges and Affected Soils On-Site		18	18	18	18	18
Sieve-and-Wash/In-Situ Vitrification		12 0	12 0	11 8	11 8	11 7

NOTES

a. Using 5% discount rate and 30 year post-closure care and monitoring period
b. Percent change in post-closure operating costs from those given in Tables 6-2 through 6-10

TABLE 6-13

PRESENT WORTH DISCOUNT RATE SENSITIVITY ANALYSIS

Arkwood, Inc Site
Omaha, Arkansas

Alternative [b]	Net Present Value of Alternatives Costs (Millions of Dollars) [a]							
	3%	4%	5%	6%	7%	8%	9%	10%
No Action	0 37	0 33	0 29	0.26	0 23	0 21	0 19	0 18
Site Monitoring and Restricted Access	0 50	0 45	0 41	0 37	0 34	0 32	0 29	0 28
Incinerate Sludges	2 2	2 2	2 1	2 1	2 1	2 0	2 0	2 0
Incinerate Sludges / Topsoil Cap Over Entire Site	3 2	3 1	3 1	3 0	3 0	3 0	2 9	2 9
Incinerate Sludges / Consolidate and Cap-in-Place Affected Soils	4 2	4 2	4 1	4 1	4 0	4 0	4 0	4 0
Incinerate Sludges / Consolidate Sieve-and-Wash and Cap-in-Place Affected Soils	6 7	6 6	6 6	6 5	6 5	6 4	6 4	6 4
Incinerate Sludges / Sieve-and-Wash Drying Carry Treat Fines and Cap-in-Place Affected Soils	$14	$14	$14	$14	$14	$13	$13	$13
Incinerate Sludges / Landfill Affected Soils On-Site	5 6	5 5	5 5	5 4	5 4	5 4	5 3	5 3
Incinerate Sludges and Affected Soils On-Site	18	18	18	18	18	18	18	18
Sieve-and-Wash/In-Situ Vitrification	$11 9	$11 9	$11 8	$11 8	$11 8	$11 7	$11 7	$11 7

[a] Using the indicated discount rate and 30 year post-closure care and monitoring period

[b] Discount rate used for present worth calculation

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TABLE 6-14

VOLUME SENSITIVITY ANALYSES
FOR SLUDGES AND AFFECTED SOILS

Arkwood (DOE Site)
Omaha, Arkansas

Alternative		Net Present Value of Alternatives Costs (Millions of Dollars) [a]				
		+150% [b] (52,100 CY)	+100% (41,700 CY)	+50% (31,200 CY)	0% (20,800 CY)	-50% (10,400 CY)
A	No Action	0.29	0.29	0.29	0.29	0.29
B	Site Monitoring and Restricted Access	0.4	0.4	0.4	0.4	0.4
C	Incinerate Sludges	4.3	3.6	2.8	2.1	1.4
C1	Incinerate Sludges / Top Soil Cap over Entire Site	5.1	4.6	3.8	3.1	2.4
D	Incinerate Sludges / Consolidate and Cap-in-Place Affected Soils	6.7	5.8	4.9	4.1	3.2
E	Incinerate Sludges / Consolidate, Sieve-and-wash and Cap-in-Place Affected Soils	11	9.6	8.1	6.6	5.1
F	Incinerate Sludges / Sieve-and-wash Biologically Treat Fines and Cap-in-Place Affected Soils	24	21	17	14	10
G	Incinerate Sludges / Landfill Affected Soils On-Site	10	8.4	7.0	5.5	4.1
H	Incinerate Sludges and Affected Soils On-Site	39	32	25	18	11
I	Sieve-and Wash/In-Situ Vitrification	17.7	15.7	13.8	11.8	9.9

NOTES
[a] Using 5% discount rate and 30 year post-closure care and monitoring period
[b] Volume of sludges and affected soils

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TABLE 6-15

Alternative 1
Sieve and Wash/Insitu Vitrification

Arkwood, Inc Site
Omaha, Arkansas

Capital and Operating Costs	Quantity	Units	Unit Cost		Cost
Common Items					$310,000
Excavate sludges	430	cy	$3 50		2,000
Excavate affected soils	20 400	cy	$3 50		71,000
Sieve and wash affected soils					1,060,000
Dike around consolidated area	1,000	lf	$71 00		71,000
Insitu vitrification	8,000	cy	$475 00		3,800,000
Topsoil cap over entire site	650,000	sf	$0 74		480,000
Backfill site	2 400	cy	$9 50		23,000
Site facilities & utilities - capital					77,000
Site facilities & utilities - operating	1	yr	$183 000 00		183,000
Restore disturbed areas	15	ac	$19 000 00		285,000
Subtotal					$6,362,000
Contractor Overhead Profit and Bonds				x	1 20
Contingency				x	1 25
Engineering & Construction Surveillance				x	1 20
Estimated Capital & Operating Cost					$11 451,600
Post-Closure Care Costs					
Cap maintenance	15	ac/yr	$150 00		2 300
Fence maintenance	5 000	lf/yr	$0 40		2,000
Annual Subtotal					$4,300
Net present value subtotal (b)					66,000
Groundwater monitoring (net present value) (b c)					194 000
					$260,000
				x	1 25
				x	1 20
Net Present Value Post-Closure Cost (b)					$390 000
Estimated Alternative Cost (net present care cost) (d)					$11 841,600

NOTES
(a) Costs are mid-1989
(b) Assumes an interest (discount) rate of 5% (net of inflation) and a 30-year post-closure care period
(c) Monitoring frequency and parameters as specified in Section 5
(d) The sum of capital and operating costs and the net present value of the post-closure care costs

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TABLE 7 1

Summary of Evaluation of Remedial Alternatives for Sludges and Affected Soils

Arkwood Inc Site
Omaha Arkansas

	Alternative	Protection of Human Health and the Environment	Compliance with ARARs	Long-term Effectiveness and Permanence	Reduction of Toxicity, Mobility or Volume
A	NO Action	NO reduction in risk from the site which risk is acceptable according to EPA guidance	Does not reduce toxicity mobility or volume of site constituents	Existing risk remains NO certainty of long-term success	No treatment or containment Does not reduce toxicity mobility or volume
B	Site Monitoring and Restricted Access	Reduced risk of direct contact by controlling access Provides for detection of migration if it occurs	Does not reduce toxicity mobility or volume of site constituents	NO certainty of long-term reduction of risk	No treatment or containment Does not reduce toxicity, mobility or volume
C	Incinerate Sludges	Destroys sludges Residual risk acceptable according to EPA guidance	Complies with ARARs	Effectively reduces risk for the long term	Destroys an estimated 40% of mass of site constituents
C1	Incinerate Sludges/Top soil Cap over Entire Site	Destroys sludges Capping eliminates risk due to direct contact	Complies with ARARs	See Alternative C Capping is effective if properly maintained	See Alternative C
D	Incinerate Sludges/ Consolidate and Cap-In-Place Affected Soils	Destroys sludges Provides containment of affected soils under composite cap	Complies with ARARs	See Alternative C1	Effectively reduces toxicity and volume by incinerating sludges Containment under composite cap reduces mobility

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TABLE 7 1 (cont d)

Summary of Evaluation of Remedial Alternatives for Sludges and Affected Soils

Arkwood Inc Site
Omaha Arkansas

	Alternative	Protection of Human Health and the Environment	Compliance with ARARs	Long-Term Effectiveness and Permanence	Reduction of Toxicity, Mobility or Volume
E	Incinerate Sludges/ Consolidate, Steve-and-Wash and Cap-in-Place Affected Soils	Destroys sludges Additional constituent removal via steve-and-wash treatment Capping eliminates residual risk	Complies with ARARs	See Alternative C1 Additional treatment enhances the permanence of the remedy Leachate testing indicates treatment performance equivalent to incineration	Additional reduction in toxicity, mobility and volume via treatment of affected soils
F	Incinerate Sludges/ Steve-and-Wash, Biologically Treat Fines and Cap-in-Place Affected Soils	Destroys sludges Additional constituent removal via steve-and-wash and biological treatment Capping eliminates residual risk	Complies with ARARs	See Alternative E Additional treatment enhances the permanence of the remedy Leachate testing indicates treatment performance equivalent to incineration	Additional reduction in toxicity, mobility and volume over Alternative E via biological treatment
G	Incinerate Sludges/ Landfill Affected Soils On-Site	Destroys sludges Provides containment of affected soils in RCRA landfill Capping rest of site eliminates residual risk	Complies with ARARs	See Alternative C A RCRA landfill is effective, if properly maintained	See Alternative C Containment provides additional reduction in mobility and effective toxicity of constituents
H	Incinerate Sludges and Affected Soils On-Site	Destroys site constituents of concern	Complies with ARARs	No maintenance or monitoring required	Provides maximum destruction of constituents
I	Steve-and-Wash/In-Situ Vitrification	Destroys site constituents of concern	Complies with ARARs	No maintenance or monitoring required	Provides maximum destruction of constituents

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TABLE 7.1 (cont'd)

Summary of Evaluation of Remedial Alternatives for Sludges and Affected Soils

Arkwood, Inc. Site
Omaha, Arkansas

	Alternative	Short-term Effectiveness	Implementability	Present Worth Cost
A	No Action	No short-term reduction of risk from the site	No capital improvements. Most easily implemented	$0.29 million
B	Site Monitoring and Restricted Access	Short-term reduction of risk by fencing	Limited work required. Easily implemented	$0.4 million
C	Incinerate Sludge	Provides a significant reduction in risk with a short-term effort	Limited site work required. Easily implemented	$2.1 million
C1	Incinerate Sludges/Topsoil Cap over Entire Site	Provides a significant reduction in risk with a short-term effort	Limited site work required. Easily implemented	$3.1 million
D	Incinerate Sludges/ Consolidate and Cap In-Place Affected Soils	See Alternative C. Slightly more worker exposure due to consolidation	Limited site work required. Easily implemented	$4.1 million

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TABLE 7-1 (Cont'd)

Summary of Evaluation of Remedial Alternatives for Sludges and Affected Soils

Arkwood, Inc. Site
Omaha, Arkansas

	Alternative	Short-Term Effectiveness	Implementability	Present Worth Cost
E	Incinerate Sludges/ Consolidate, Sieve-and-wash and Cap-in-Place Affected Soils On-Site	See Alternative C. Short-term risk due to worker exposure during the relatively long treatment period	Requires construction and operation of a full-scale treatment plant using innovative technology.	$6.6 million
F	Incinerate Sludges/ Sieve-and-wash, Biologically Treat Fines and Cap-in-Place Affected Soils	See Alternative C. Short-term risk due to worker exposure during the relatively long treatment period.	See Alternative E Biological treatment more difficult than sieve-and-wash alone	$14 million
G	Incinerate Sludges/ Landfill Affected Soils On-Site	See Alternative C. Provides additional containment by landfilling affected soils, but increases worker exposure.	A RCRA landfill is readily constructed	$5.5 million
H	Incinerate Sludges and Affected Soils On-Site	Short-term risk during the relatively long remediation period due to handling affected materials and toxic combustion by-products	Most complex alternative. Requires construction of a very complicated plant and additional permitting and reporting efforts.	$18 million
I	Sieve-and-wash/In-Situ Vitrification	Short-term risk during relatively short remediation period due to handling affected materials and toxic combustion by-products. Total emission control.	Moderately complex alternative. Required mobilization of trailer mounted equipment, trained personnel and modification to electrical service	$11.8 million

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TABLE 7 2

Summary of Comparative Analysis
Remedial Alternatives for Sludges and Affected Soils

Arkwood, Inc Site
Omaha Arkansas

Alternative	Protection of human health and the environment	Compliance with ARARs	Long-term effectiveness and permanence	Reduction of toxicity, mobility and volume	Short-term effectiveness	Implementability	Time to Completion (years)	Cost (millions)
A - NO ACTION	--	.	--	--	--	++	--	$ 0 29
B Site Monitoring and Restricted Access	-	.	--	--	--	++	0 3	$ 0 4
C Incinerate Sludges	+	+	+	+	+	++		$ 2 1
C1 - Incinerate Sludges/Topsoil Cap over Entire Site	+	.	+	+	++	++	0 5	$ 3 1
							0 5	
D - Incinerate Sludges/Consolidate and Cap-in-Place Affected Soils	+	+	+	+	++	+	1 0	$ 4 1
E - Incinerate Sludges/Consolidate Sieve-and-Wash and Cap-in-Place Affected Soils	++	.	++	++	+	-	2 0	$ 6 6
F - Incinerate Sludges/Sieve-and-Wash Biologically Treat Fines and Cap-in-Place Affected Soils	++	.	++	++	.	--	4 0	$14
G - Incinerate Sludges/Landfill Affected Soils On-Site	+	+	+	+	++	.	2 0	$ 5 5
H - Incinerate Sludges and Affected Soils On-Site	++	.	++	++	.	--	3 0	$18
I - Sieve-and-Wash/In-Situ Vitrification	++	.	++	++	+	-	2 0	$11 8

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APPENDIX G

APPENDIX G

Transcript of February 12, 1990 Public Meeting in Omaha, Arkansas

008942

Public Meeting
Omaha Public School
Omaha, Arkansas

February 12, 1990

Mr. GARRETT BONDY, Chairman

ALSO PRESENT: MR. BRENT TRUSKOWSKI
EPA
Remedial Project Manager

DEBBYE L. PETRE, CCR
PETRE'S STENOGRAPH SERVICE
207 Spring Street
Little Rock, Arkansas 72201

008943

P R O C E E D I N G S

* * * *

MS. GREENEY: The purpose of our meeting tonight is for us to discuss the remedial investigation that was done out there at the site and to share the results of that information with you. It is going to be real informal, so if you have some questions, let us know.

And we want to make sure that everybody has a blue sheet. That is the fact sheet that we are going to be talking about tonight. It has the current information on the front, with some background information, just some general information on the back.

Also, the pink is a questionnaire to let us know how we are doing, what else we can do to provide more information to you in case you are not getting enough, or if what you are getting, you don't understand, please let us know, because we would like to share the information that we have with you.

And also, the yellow card, if you will fill that in and let me have it before you leave, that will keep our computer mailing list updated

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and current.

I want to introduce tonight Garrett Bondy on the far end; he is the section chief for the enforcement Superfund sites that are in Arkansas and Louisiana. And Brent Truskowski, who is the project manager for the Arkwood site. So, I am going to turn you over to these two gentlemen to explain to you what we have up here tonight.

MR. TRUSKOWSKI: I am going to do the bulk of the presentation and try to let everybody know what is going on. As I am going, a lot of times I will just kind of skip over a point or something like that, I don't do it intentionally, it is more from just going too fast for my brain to keep up with me. So, if there is anything during the presentation that you don't understand, or you want me to explain a little bit further, please ask and just go ahead and pipe up, and I will try to explain a little better, because sometimes I will get going and tend to talk like you are talking to people you work with, which is a really difficult -- so let me know.

So, I am going to go through the history

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of the site, first, and then I am going to tell you about the sampling we did, and why we did what sampling we did at the site, and then I an going to tell you what results came from the sampling, going through a little bit about what health threat there may be, and then where we go from here, what the next few steps are.

I am going to take off my jacket if that is okay with everybody. It is a little warm in here today.

The site, as we know it, came into being about 1960, when the railroad was punching their line through, they used the site to get material for embankments. They just went in, they ripped out this flat area out of the hillside. In 1962, a company called Arkwood, Inc. started up wood treating with a single cylinder wood treatment facility, a pressure cylinder, and their major constituent was pentachlorophenol, PCP, carried with diesel fuel. In 1973, Mass Merchandisers, Inc., bought out Arkwood, Inc., and operated the site until 1984.

In 1981, the first complaint came about some potential contamination the site. Where that came from was in the railroad there is a

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spring that goes out of the railroad tunnel, and when they were driving the train through, and they were working there, they complained of a real chemical odor, which ADPC&E investigated, and it turned out that it was pentachlorophenol coming out from one of the springs.

At the same time to help understand what was going on, M.M.I. hired McClellan Consulting Engineers to try to help them find out what was going on with the ground water. In 1985, they hired Geraghty and Miller, and in 1985 also they hired McKesson Environmental Services to try to help out a little further to understand what was going on.

They closed down the site in 1984, and then after the site was proposed for the National Priorities List, which is what the Superfund money is used to clean up, M.M.I and E.P.A. entered into an administrative order of consent under which MMI would perform the remedial investigation feasibility study with E.P.A. oversight. In 1987, ERM-Southwest took over the project for MMI.

I want to go into real quickly what a remedial investigation/feasibility study is. In

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the Superfund process, E.P.A. will identify a site at first, and then go in and do the preliminary testing to try to get a site onto the National Priorities List. After it is placed, or it is proposed, we go and do a detailed investigation of the site, which will give us a feel for where the contamination is, how much contamination there is, where the contamination is going, and what the health threats from the site may be, after which we will do a feasibility study to find out what the best alternative may be for any given site. After that, we select a remedy and I'll get into that part later and the site future part of it.

From the sampling that we did, the Arkwood site used pentachlorophenol carried by diesel oil, which is called a polynuclear aromatic hydrocarbon, called PNA, if I start lapsing into that, using that terminology.

And basically, the way the site operated is the treating cylinders were right in here, in the plant location, the plant building, and then after that, they rolled it out onto railroad tracks to let it drip out, and then they take it over here and go ahead and store it in the

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storage yard. Because of what we knew about how the site operated, we decided that the places that most likely contamination would be about right in here to about right in here, from the treatment area out to the storage yard. There is also an ash pile that was created by, I don't know, a burner, a furnace that they had on-site, and they dumped all of the ash out here. And there is also, of course, a sink hole where they dumped wastewater from the cleaning out process, they just threw it in there. And then there is also what we call the railroad ditch area, which is another place that they disposed of the wash water from the site.

So, we what we did first M.M.I. and EPA set up a grid sampling over the entire site, a large grid sampling in degrees of about 50 foot centers, 50 foot from sample to sample, trying to get a feel for where exactly the contamination is on the site. Then once we decided where there was approximate contamination, we went in and did a fine grid sampling to really delineate it and try to get a feel for exactly how much there was.

We also did a sediment sampling for rain runoff, for runoff through this ditch here going

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into the railroad tunnel, and up here, where Cricket Spring is, off of there. Then we also looked in the area for drinking water wells that may be potentially contaminated, and springs, like New Cricket Spring. That may be potentially contaminated, and selected wells and springs for sampling, also.

And then on-site, we also sunk some monitoring wells to try to get a feel for where the ground water was moving, and what the ground water did once it got underneath the Arkwood site to try to get out.

The results of the sampling turned out pretty much like we expected. The highest areas of contamination of the soil being right here where they did the actual treatment, and then where they wheeled it out to drip, and then out in the wood storage yard where they were put out to dry. We ended up with some hot spots right in here, right in here, with some more median areas right in here and right in here. The railroad ditch area we also found is a place of high contamination, also pretty much as expected, and then most of the rest of the site is contaminated with low levels. Some sampling turned out to

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have very low levels, you know, a little bit of slow washing off the site, but very, very little. Next to the railroad ditch, we have the highest levels of contamination of pentachlorophenol and (Inaudible).

Yes, sir.

EDSEL BAKER: What about those wells you drilled there, how deep did you go?

MR. TRUSKOWSKI: We went anywhere from, I think about 20 feet down to about 80 feet. We went through a couple of different units, there is a top geologic unit, which is just the soils.

EDSEL BAKER: What was your deepest site, well site?

MR. TRUSKOWSKI: The deepest one we have is a well that was actually already there; it is down a little over a hundred feet. And then we put some deeper ones in the railroad ditch area.

EDSEL BAKER: What did they show?

MR. TRUSKOWSKI: The deep ones didn't show any contamination.

EDSEL BAKER: The shallow ones showed the most contamination?

MR. TRUSKOWSKI: Right. The ones that were just in the dirt, in the top layer of dirt, down

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to the first real rock layer.

EDSEL BAKER: You mentioned Cricket Springs, you said that was contaminated. What was the extent of that?

MR. TRUSKOWSKI: Sir?

EDSEL BAKER: What was the percentage of Cricket Springs that was contaminated?

MR. TRUSKOWSKI: The percentage would be about --

MR. BONDY: Two parts per million.

MR. TRUSKOWSKI: What is that, about .02 percent, is that how it translates across?

EDSEL BAKER: That all run down Cricket Springs?

MR. TRUSKOWSKI: Right, right, it comes out -- it comes out of Cricket Springs along the channel, and this water disappears and comes back, and disappears and comes back, and eventually runs into Cricket Creek.

EDSEL BAKER: There is a sign on the other side of the road there that says water coming out of that cave is contaminated. What is the story there?

MR. TRUSKOWSKI: What the remedial investigation shows as far as the contamination

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of that creek goes, where the spring is, is the level is just above what the E.P.A. calls drinking water criteria, which is if you are going to be drinking it, say the public system, and it had just over the drinking water criteria, then we'd say it is not safe to drink because of this, but it is not very much above it at all.

EDSEL BAKER: What is the government's combination, when they had a big run-off like they did the other day?

MR. TRUSKOWSKI: We haven't got those results back. I wish we had. I am very curious, too. We pushed real hard to have it done when you're waiting for that kind of rainfall to get it, the problem is is that when we take a sample, send it off to the lab, and the lab takes 30 days to get the results, we just haven't got them yet. They will be available in the final remedial investigation.

WILLIE TATE: Has the level went down continuously since you first monitored?

MR. TRUSKOWSKI: Yes and no. It went down for a while, and then it came back up slightly. We are not -- one of the problems on the site that were shown up in the drilling of the

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monitoring wells is -- I don't know if you are very familiar with the geology of the area, the caves and the Karst aquifers is what we have got, and the big problem with that is that you just don't know really where things are going. What happens is it goes down, then it will dilute out, or dissolve out a cave, a solution channel through the rock, so that when you come to drill wells, which is our usual way of getting a good feel for what is going on in the ground water, it is a hit and miss type thing, because you have got just like the caves, the springs coming out of the road cutter, the Cricket Spring, where it comes out of a cave about that big, is that if you have got a cave this big and you drill right here, you have missed it. It is like throwing darts at a dart board. You put a map up and you throw darts at it, and that is as good a possibility of finding one of these channels as anything.

MRS. ADAMS: Would a dye study show this?

MR. TRUSKOWSKI: We have talked about it. We are going to do one as part of phase two monitoring. We did a phase one, and then a phase two, the problem is the drought, drought

conditions that really, it really has kept us from doing some things that we really wanted to do. We are still considering doing dye tracing studies as part of the remedial design to decide where the water is going. We are going to have to see what the results of our -- that latest sampling, the high water sampling turns up, to see if we really want to do it or not, and weigh it against what we would get out of it.

EDSEL BAKER: You haven't sunk a test site below the Arkwood site, have you?

MR. TRUSKOWSKI: No, you mean a monitoring well outside the site boundaries? Just along the road down in here, down this area here.

EDSEL BAKER: I mean, on down the creek.

MR. TRUSKOWSKI: You mean down over here?

EDSEL BAKER: Yeah.

MR. TRUSKOWSKI: No, we haven't done any wells down there.

EDSEL BAKER: Are you going to?

MR. TRUSKOWSKI: No.

EDSEL BAKER: Why?

MR. TRUSKOWSKI: Okay. We have got the top ground water zone, which is contaminated, and it goes down and it hits the sandstone unit, that

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doesn't let the water through, and then we have got the lower units. It happens that Cricket Spring represents where the ground water comes out at that area, that is the top of the Sylamore, the sandstone unit that I am talking about, and then after that, it is just not there, the contamination comes out there.

EDSEL BAKER: Am I right in assuming that Mass Merchandisers bought the place across the road because of the levels of contamination, is that right?

MR. TRUSKOWSKI: I'm not sure what exactly the reason why M.M.I. bougnt it. I heard that they did, but I don't know the details of it.

EDSEL BAKER: I was just curious how deep that well was, and how come to get in there.

MR. TRUSKOWSKI: I don't know, I really can't say. It hasn't turned up --

EDSEL BAKER: That means every well is suspect, doesn't it?

MR. TRUSKOWSKI: Sir?

EDSEL BAKER: That means every well in the area is suspect, then, doesn't it?

MR. TRUSKOWSKI: Well, yes and no. You know, we tested the wells afterward, that same

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well afterwards, and nothing has turned up there.

EDSEL BAKER: How many wells did you test in this area?

MR. TRUSKOWSKI: I think we tested somewhere around ten.

EDSEL BAKER: Very few, isn't it?

MR. TRUSKOWSKI: I really can't -- it depends on how the site --

EDSEL BAKER: It is damn few if you live around here.

MR. TRUSKOWSKI: Sir?

EDSEL BAKER: It is damn few for the people that live around here.

MR. TRUSKOWSKI: Right. Again, I have got to point out that there hasn't been anything show up in any of the wells sampled.

KEN ADAMS: I live below (Inaudible.), and the first three days rain we had washed down here at the water.

MR. TRUSKOWSKI: The last one?

KEN ADAMS: The first one.

MR. TRUSKOWSKI: Oh, okay. The one about two years ago.

KEN ADAMS: No. There have been two big heavy rains since then. And the first rain that

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we had, we went out, and I could smell diesel, smelled like diesel fuel going up through it.

MR. TRUSKOWSKI: Yes, we noticed that today.

KEN ADAMS: Okay. My well is behind the house, and you are saying that Cricket Spring is above that, that is going down the creek, that goes into my well, and how do you explain the sandstone level there? You are going to have about three or four different stages, and you can't always say there is going to be a sandstone level in between, because you are going to have grass, plus you have got the railroad up there with the dirt, the railroad ties that they have towed off and contaminated with the same stuff, and it lays around, plus then you have got the runoff that runs down into the Cricket, which runs into Table Rock Lake, now how are you going to explain the contamination there?

MR. TRUSKOWSKI: Well, the pentachlorophenol, in this environment, what happens is the sunlight, the UV breaks it down in the water itself, so as a result, downstream a hundred yards, the penta is gone. It just isn't there any more, that is what is happening in the creek channel itself. So, at the time it is

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actually down to Cricket Creek, it is just not there any more. And so, by the time it gets to Table Rock, Table Rock Lake, it is gone, there is nothing.

WILLIE TATE: In the first testing, (Inaudible.), and the contamination coming off of (Inaudible.) the -- can't you find anything (Inaudible.)

MR. TRUSKOWSKI: This latest sampling, you mean? I haven't got the results back, I don't know. That one's been turning up clean in the entire investigation, also.

WILLIE TATE: I don't know if there is --

MR. TRUSKOWSKI: Yes. We were noticing that today, we walked down that way and smelled that same smell of diesel in there, and I don't know where it is coming from. You know, when you smell it at the spring, itself, and that is probably, you know, what is stuck to the rocks, from many years of coming through. But the level of penta, you know, the thing that you really have to worry about in the wells, it is approaching a level that we don't really need to worry about.

EDSEL BAKER: What about the erosion? When

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you have got the erosion, all that soil that is contaminated at the Arlwood site, it is still going to get into the water system and it is going to be there.

KEN ADAMS: The water table, if you don't know where it is going, all of our water could be contaminated, too, you don't know which way the water is going, what the water table is, or anything like that.

MRS. ADAMS: Our concern has been in the sinkhole area. We were told that they actually dumped the sludge, not just the wash water, and, of course, it being an underground system in the rock areas down there, that has been our concern and we haven't heard any word on that testing.

MR. TRUSKOWSKI: It is something that we are real concerned about, also, going into the investigation, the reports of the sinkhole. As a part of the -- in the phase two, we actually took the concrete cap off the sampling material that was at the site, and so, something that really shocked everybody involved is that it turned up fairly clean. So, there wasn't anything in there. There is nothing in there. The sediment sampling that we did on the runoff locations

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also, we didn't find anything in the sediments, either, in the spring channel, or from Cricket Creek, or even going through the railroad, in the railroad ditch, going back here this way, this being one of the most logical places for it to go, you've got a real high level of contamination right here. You'd think the runoff would go through here, but it didn't show up there, either.

MRS. ADAMS: (Inaudible.)

MR. TRUSKOWSKI: Right. That means we didn't detect it. You know, any time you do a lab sampling, you have a limit to which the machine will go, and, you know, it may be present and it may not be below that, but it wasn't present in the level that the machine can read.

MR. BONDY: What about the results of the sampling (Inaudible.) The point is we're not finding this stuff off site.

MR. TRUSKOWSKI: The only place it is showing up off site is in Cricket Springs, which, like I said, is rapidly approaching very near drinking water standards, which is really a very good sign for us, because when we first started investigating the site, the levels were elevated

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to a point that we probably would have some pretty big concerns about, and if we still had that level now, we'd probably want to set up some kind of treatment system, which we may still. We haven't ruled that out. But the levels have come down to a point where they are very near drinking water standards, which means that something is going on. Maybe that is because the site does not operate any more, they're not putting any more penta in the ground, or maybe the stuff is just draining naturally, but it is not showing up in the concentrations that it once was.

JUDY BOYD: That is how it is not showing up in the --

MR. TRUSKOWSKI: I'm sorry?

MRS. ADAMS: That is how it is not showing up in the -- (Inaudible.)

MR. TRUSKOWSKI: I think we have got about ten. I don't remember the exact number.

JUDY BOYD: Do you have any idea like what percentage of the total wells in the area that would represent?

MR. TRUSKOWSKI: How big an area?

JUDY BOYD: Well, you know, if we encompass, say, the -- like four -- well, let's limit it to

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even two square miles, to Omaha town and two square miles around it.

RAYFORD DUGGAN: The city well represents all the town, and there is nothing ever been really found in the city well. It was tested -- (Inaudible.)

MR. TRUSKOWSKI: Right. And as far as the private wells go in I would say two square miles, two mile by two mile square region, I think there is something like 40 wells in the area. Again, I am doing this all from memory, looking at the maps that we have generated as part of the survey, well survey. We ran into a problem there with a lot of the wells that have been drilled and been put in being used as just spring wells, things like that. The way we found what wells we found was going through the county, to the city, and a lot of the wells may or may not be registered with the county or the city. So, that's the ones that we have. So, I would say 40, maybe 50, which would give us 25, 20, 25 percent of the wells. Take that with a grain of salt because I am just running that from memory.

KATHY DUGGAN: How long ago were those wells tested?

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MR. TRUSKOWSKI: I think the last sampling event we did was the wells a few months ago. Two months ago, does that sound right?

KATHY DUGGAN: No, it was about three weeks ago, as a matter of fact.

MR. TRUSKOWSKI: During the high flow sampling?

MR. TRUSKOWSKI: The ones three weeks ago, we are still waiting on the results. The ones three months ago echoed the same results that we found all along, which was that nothing was showing up.

KATHY DUGGAN: I would like to see a copy of my results. We have been having -- (Inaudible.) -- trouble getting the results back to us. The last ones that we got were in June of '89.

MR. TRUSKOWSKI: Those are the ones that I had and had Patty send to you.

WILLIE TATE: Did you ever find any pentachlorophenol in any wells?

MR. TRUSKOWSKI: During the original investigations, when we started the investigations, there was a well, one well that turned up something. But since then, there's been nothing. Let's see. I think I have beat

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the monitor wells to death, the ones we've done on the site, but the thing about the monitor wells that we did drill on the site in this area and down in this area, is that they did show contamination, and in levels that were higher even than what we were getting at Cricket Springs. So, what that is telling is that these wells here, the contamination is there, and then it is going down and the other ground water is coming in and diluting down to the level that we see near Cricket Springs. But there is a problem with the Karst geology in the area, too, which is really throwing a wrench into the investigation.

During the remedial investigation, we also did what was called an endangerment assessment. What it is designed for is to give us a feel for what the health threats from the site would be. And the majority of the health threat from the site . from this area right here, the most highly contaminated area right here, from the railroad ditch area, where we turned up fairly high levels of -- actually, very high levels of pentachlorophenol, I believe high levels of PNAs, and some low levels of dioxin furans. And this area represents the highest

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threat on the site.

Another area that turned up to be -- (Inaudible) -- a health threat is this area right here, the treatment area, the drip out area, but again that is because that is the highest level of contamination, so, of course we included it. The rest of the site really doesn't pose much of a threat at all.

MR. BONDY: Except for the hot spots.

MR. TRUSKOWSKI: Yeah. The hot spots in here and then there's a spot in here that will pose a health threat. For the majority of the site a health threat really isn't that high, especially as when you compare it with other Superfund areas in Arkansas.

Cricket Springs, as I said before, the major health threat there is the drinking water criteria. If you were to drink the water, you would be drinking water that was contaminated slightly above what E.P.A. considers to be an acceptable level of pentachlorophenol.

KATHY DUGGAN: How acceptable do we all really know about the levels of the stuff?

MR. TRUSKOWSKI: I'm not sure.

KATHY DUGGAN: I mean, what is their

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acceptable level and how do we know?

JOHN PARTON: What percentage would it take to make you alarmed, to really get on the ball?

MR. TRUSKOWSKI: In the water?

JOHN PARTON: Yes.

MR. TRUSKOWSKI: The level that we have got it at, just over the drinking water criteria, it does set off bells in our head. But where that is coming from, that we reckon it is coming from, is getting the water percolating through the areas of high contamination, the railroad ditch, from these hot spots here, and going down into the site into the drinking water -- not the drinking water, I'm sorry, to the creek, or the spring. What we are reckoning on is we are still considering putting in a drinking system at that well head, where the spring comes out of the ground. And we are also reckoning on when we remove the gross source of the contamination, because the contamination isn't there any more for it to leach through when it is rained through, that level would even back off some more. So, a significant decrease in the levels when the site stopped operating, and now we think we will see another one when the site is cleaned

up.

JOHN PARTON: The last time you were up here, you said that what once you got on the site, it would take about a year and then you would really get down to business. And you have had a year to really examine things, and we are kind of waiting for somebody to get down to some business.

MR. TRUSKOWSKI: You mean, like cleaning up the site?

JOHN PARTON: Yes, doing something, yeah.

MR. TRUSKOWSKI: So would we. We would really love to get it done in the next year, if we could.

JUDY BOYD: When you say that you are still considering setting up a treatment facility for the well --

MR. TRUSKOWSKI: For the spring.

JUDY BOYD: For the spring, okay. What criteria would you use to determine whether or not you will put in a treatment facility?

MR. TRUSKOWSKI: That is a very good question. We haven't decided that yet. We are still -- we just got the draft of the remedial investigation feasibilty studies in.

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MR. BONDY: What we look at is to see how much danger it represents to the public health.

JUDY BOYD: Well, it will be determined by the level of toxins present in the water?

MR. BONDY: And the likely exposure of people. It is not based on numerical criteria, on the drinking water standards, one part per million, for example, just because it is above one part per million, we will not necessarily put in a treatment plant, because it is not used as a drinking water source.

JUDY BOYD: Will input from the community affect that provision at all, as far as them putting in a treatment plant?

MR. BONDY: Yes.

MR. TRUSKOWSKI: It very likely could.

KATHY DUGGAN: When you talk about health hazards, what are you talking about, the amounts?

MR. TRUSKOWSKI: Penta? I don't believe so --

MR. BONDY: I don't know. We are not really qualified to answer the health effects in great detail; you need a toxicologist type person. We can get back to you on the health effects question.

KATHY DUGGAN: That is what I want to know,

when they say how much is acceptable parts per million, how do they know that? They don't know that.

MR. BONDY: Right. We can't, we are not -- the basis for that number I couldn't tell you.

KATHY DUGGAN: Who decided that?

MR. TRUSKOWSKI: Yes, you need our staff chemist. The major threat from the site actually isn't from the pentachlorophenol, it is from the diesel that they used to carry the PNAs, that they used to dissolve the pentachlorophenol in the diesel and then to pressure treat the wood with. And part of the formulation in the diesel is some carcinogenic, cancer causing agents, and penta isn't really what is driving the health threats so much as the PNAs.

MR. BONDY: Let me clarify one thing. We have been talking about the pentachlorophenol because that is the most common thing out there, so we use that as an indicator to chase everything else. So when we see pentachlorophenol, we think there might be other things that would be there with it. That is why we keep talking about the penta, because that is the most common thing that's the easiest thing

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for us to see. Everything out there -- everything else out there is cut there to a much lesser extent.

MRS. ADAMS: You said something about dioxins and sulfacants?

MR. TRUSKOWSKI: Dioxins and furans?

MRS. ADAMS: What is that? Isn't dioxin one of the most dangerous things we make?

MR. TRUSKOWSKI: Yes. Depending on the exact formulation of the dioxin.

MR. BONDY: There are many, many different kinds of dioxin. The one that you referred to --

MRS. ADAMS: Is not this one?

MR. BONDY: It is not this one.

MRS. ADAMS: Is it related to this one?

MR. BONDY: It is related. There is a big family of chemicals, they are all very similar, and it depends on how many chlorine atoms are attached on the molecule, and where they are located on the molecule, and all of that is very, very significant for the health effects. What we do then is we do the analysis to show you what kind of dioxins you have out there, and then there are multipliers, factors that you apply to the concentrations, and you add them all up and

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that gives you an equivalent of the worst kind. You reference everything back to the worst kind. So, if you had so many parts per billion of the worst kind equivalent at the site, you multiply that to get a comparison if you had that kind how bad it would be.

MRS. ADAMS: What does all that mean about how bad it is?

MR. BONDY: Brent, the levels were?

MR. TRUSKOWSKI: The levels were about, what, one in 10,000?

MR. BONDY: No, we are talking about dioxin.

MR. TRUSKOWSKI: Oh, about 14 parts per million.

MR. BONDY: Fourteen part per million is the highest we saw.

MRS. ADAMS: What does that mean?

MR. BONDY: What that means is the acceptable standards for if you were in your back yard, your children were playing every day out in the back yard, you would have one.

JUDY BOYD: Is that the same for drinking standards?

MR. BONDY: Drinking water standard, no, I don't know what the drinking water standard is.

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JUDY BOYD: Is it less?

MR. BONDY: It would be much, much lower. We were't even finding it in the water. It is very insoluble, so you tend not to see it in the water; it attaches to the sand particles. But in terms of the dioxin action levels, one part per billion is the accepted levels where if children were playing with it in the back yard on your lawn. The standard, there is a less accepted level for what is okay for industrial use as far as, say, if you were to go to work and you were working in this yard that had dioxin in the soils, the typical guide that you hear is around 20.

KATHY DUGGAN: That was 14.

MR. BONDY: That was 14, and that was the absolute highest that we saw. Typically, we would only see ones or twos, nothing usually, but we had some ones and twos.

EDSEL BAKER: If we don't know where the surface water is going, where is the ground water going?

MR. TRUSKOWSKI: It is pretty much flowing to the west. It is flowing from here off this way which puts it right about here.

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EDSEL BAKER: Where is it going down Cricket Creek.

MR. TRUSKOWSKI: Yes and no, when it comes out, it comes out on the surface, it comes out in Cricket Spring. When it is underground, it is moving approximately this way, approximately to the west. Right in like this, which is why we were seeing it in Cricket Springs, and say, nowhere out in here, or out over here or up in here.

EDSEL BAKER: It makes the place bought by M.M.I. across the hill over there, that would be in line.

MR. TRUSKOWSKI: Right. But again, I don't know --

EDSEL BAKER: We are talking about ground water now, right?

MR. TRUSKOWSKI: Right. I don't know the details of why they bought it out. What I heard was that they bought it out because the contamination showed up.

EDSEL BAKER: What does the geologist say about the ground water?

MR. TRUSKOWSKI: In what respect?

EDSEL BAKER: You said west, we know it's

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going west, we're taking your word for it.

MR. TRUSKOWSKI: That is what the remedial investigation showed up. The project manager for ERM-Southwest working for M.M.I. is a geologist registered here in Arkansas. Our oversight contractor project manager is also a geologist registered here in Arkansas, and they tend to share the same view about what the ground water is doing, approximately. It is hard to say absolutely.

EDSEL BAKER: And given a few years time span when it soaks down into the ground water, then what?

MR. TRUSKOWSKI: Then we have the same situation we have now. That is what has been happening ever since 1962, that is, with the operations of the yard, there tended to be -- (Inaudible) -- down coming out. We had one spring show up about right here, in the railroad tunnel, with penta. That was the one that prompted the investigation of the site. Then we had Cricket Springs show up also. And once the operation stopped, we never found anything again here in this one. What we figured was happening was the treated wood was drying here and dripping

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here and coming down and going out of that spring. Now that everything is gone, this one has stopped, and what is happening is leaking down into the problem areas, here and down in here, and going out here.

EDSEL BAKER: What would happen in the future when you finish whatever it is you're going to do there and find out there was ground water contaminating the ground water?

MR. BONDY: Then, first off, part of the remedy will include ground water monitoring for many, many years, like 30 years, so we will continued to monitor the area of the ground water. And then there is always -- there is always a place for us to come back out here if there is a problem.

MR. TRUSKOWSKI: The future of the site. We should have the final reports ready for you to look at at the end of April -- I'm sorry, the beginning of April.

RAYFORD DUGGAN: If our well -- how do we know that when you are not cycling -- that this is not going to run in periodically, how do we know that? Water, you know, go down and flush it out, and the water runs down --

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MR. TRUSKOWSKI: Right. Hopefully, with the, you know, Garret said that we would keep monitoring the wells and the springs for probably 30 years. If something shows up, then we have to take further action.

RAYFORD DUGGAN: I'm not supposed to be drinking this water all this time? I mean, if it showed up once, what is to keep it from doing it again?

JUDY BOYD: And what level have you set at which you said you would do something?

MR. TRUSKOWSKI: We haven't set the level yet.

JUDY BOYD: You haven't set that criteria.

MR. TRUSKOWSKI: There is no criteria in place, yet.

MR. BONDY: The drinking water source is, I think, the action level is one, that is a drinking water standard, but that is a different story from the creek we were talking about earlier, which is not a drinking water source. Now, in terms of whether it is flushing in and out of the well, that is what we were shooting for in the last round of sampling to see when we did get a big rain to see what has happened. So,

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we try to sample under different conditions to see what happens when it is dry, to see what happens when it is wet.

KATHY DUGGAN: I guess our concern is that the results have been notoriously slow in getting to us, and, you know, meanwhile, we have had some problems with burning eyes. I took my son to the ophthalmologist last week, and they said, well, you know, possibly allergies, red eyes, (Inaudible), burning, things like that. You know, the thing being is, we don't have any assurance if it is allergies or this, or, you know, whatever, and of course, we are concerned. We have got two children over there drinking this water daily.

MR. BONDY: I understand your concerns.

RAYFORD DUGGAN: That is not something that can be taken care of next week or next year, but needs to be done now.

JUDY BOYD: Was the water here at the school tested?

MR. TRUSKOWSKI: The school come off the city supply, and the city supply is tested regularly.

RAYFORD DUGGAN: When it showed up, they

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didn't tell us until after another sample was taken, and why was that?

MR. TRUSKOWSKI: I don't know. I really can't say. You know, (Inaudible), it wasn't my site at the time, but I really don't know what happened earlier, the mechanics of when you got the results, when they sent them out.

RAYFORD DUGGAN: There have been a lot of discrepancies in the whole sampling.

MR. TRUSKOWSKI: Well, let me give you a call tomorrow, and I will see if I can track down all of the sampling. And, you know, a lot of that is as much my fault as anything else. We have talked on the phone about -- I sent you something in June, right? Anyway, let me get your name and number after we are done, and I will talk to you.

LADY: There are many.

MR. TRUSKOWSKI: Right.

KATHY DUGGAN: July 14th, February 23rd sample, and the results since February 23.

MR. TRUSKOWSKI: Right.

MRS. ADAMS?: (Inaudible) -- taking place, why should we -- (Inaudible.)

MR. TRUSKOWSKI: A lot of that would have to

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do with where the well is. When the investigation started, when we did the well survey, we went around to a lot of the people with wells that were on the books, talking to them, trying to get details about the construction, how they were finished, to see where they would be monitoring and if it would be worthwhile monitoring that well, depending on where it was getting water from. And that is in the (Inaudible), and they decided on the wells that they did sample.

JUDY BOYD: Did you say that you did or did not do a dye tracing sampling to know for sure about the ground water?

MR. TRUSKOWSKI: We haven't yet. We haven't yet. It is still on the -- yes, it is still an idea for the remedial design after the record is in. What the problem has been is that the amount of water that is flowing out of the springs is so low that it wouldn't tell us anything if we did it now. We need to get the springs back up to a level where it is approximately normal, rather than in this drought condition, this semi-drought condition, which it still is. I think that last year, this area was something like ten inches

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below normal in rainfall. I don't know.

If we get the conditions necessary, yes, by all means we will do it, because that takes out a lot of the uncertainty for M.M.I., who is going to be doing it, who's going to be paying for this work, and it takes out a lot of uncertainty for us, where we can come in and say, "Yeah, we definitely know where this water is going once it leaves the site."

JUDY BOYD: At what point does E.P.A. begin to pay?

MR. TRUSKOWSKI: If M.M.I. decided not to, or if M.M.I. decided not to do it according to E.P.A. guidance and policy and procedure. They have been very cooperative so far, and we haven't had really any problem with them at all. But we don't think that E.P.A. is going to have to pay for this; we think that M.M.I. is going to. Of course, we will have to see (Inaudible), they have been very good so far.

MRS. ADAMS: But you're not drinking water from the wells and worrying about it. You've got a lot more patience.

MR. TRUSKOWSKI: That is true.

MRS. ADAMS: That makes a big difference,

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when every time you make a pot of soup, you worry about what you're drinking.

MR. TRUSKOWSKI: Yes, unfortunately, the wheels of the government can sometimes be very slow, and it is frustrating to us, but it couldn't possibly be as frustrating to us as it is to you. But it is frustrating. I don't think there is anybody at the agency who didn't wish that we could just go now and take care of all of these things.

MRS. ADAMS: Why can't you?

MR. TRUSKOWSKI: Because we have to make sure that we are --

MRS. ADAMS: What can we do to speed up those wheels?

MR. TRUSKOWSKI: Write your congressman.

MRS. ADAMS: It doesn't work.

MR. TRUSKOWSKI: Yes, I know. E.P.A. is under a lot of constraints to protect the human health and environment, and these constraints are such that we have to be very careful. We are a very conservative agency. So, we have to be very careful what we are doing and how we do it, because, you know, we are in the public eye so much. As a result, we tend to study things a

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lot, a lot. And if we could speed it up, we are -- we were talking earlier, we have started doing things to speed up, we have been told to speed things up, and we are trying to put the policies in place that will allow us to speed things up, but the agency moves slowly.

WILLIE TATE: What is the lowest level that you pick up in the water? You said above one wasn't drinkable, but how far have you gone, what would be in there, and would it pick it up, or what?

MR. TRUSKOWSKI: I believe the detections are something like .01, when I was talking about just over one, I think it is around .01. In water samples, you have got to --

WILLIE TATE: If there is any PCP in the well, would it show up?

MR. TRUSKOWSKI: No.

WILLIE TATE: Not even readable in any well?

MR. TRUSKOWSKI: I don't believe so, no.

BILL NAIL: You have got (Inaudible) on a well, what is that?

MR. TRUSKOWSKI: The company that was working for M.M.I., and then --

BILL NAIL: They hired them?

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MR. TRUSKOWSKI: That is why we do split samples. I think we do about ten percent. It is a random thing, so that they don't know when we are going to split their samples. We will go out and we will split the samples and we will send them to the E.P.A. lab to check to make sure, to check the numbers back and forth, and so, yeah, they are very comparable.

Okay. But after the remedial investigation and the feasibility studies come out final, we will be putting them in the repositories. This is one repository, right? What are the other ones?

MS. GREENEY: It is on the back.

MR. TRUSKOWSKI: On the back of the sheet?

MS. GREENEY: The Boone County library, and also the Boone County courthouse, as well as the library here at the school.

MR. TRUSKOWSKI: And you will have a chance to go ahead and read those, and be able to -- if there are any questions you have about that investigation, then give me a call, you know, I will try to answer your questions the best I can. Then shortly after that, we will be putting out a proposed plan.

BILL NAIL: I want to know, I live down below all of these sites, I have got an 80 foot well about a hundred yards off of the Cricket, and to my knowledge, it has never been checked. This is where the railroad runs off, this is where your Cricket Springs runs off the railroad, right off into my place.

MR. TRUSKOWSKI: You are down below Cricket?

BILL NAIL: Just below the old Miller flag place.

MR. TRUSKOWSKI: You are down below the -- you are down in the channel, down off the channel in this direction?

BILL NAIL: Well, right down on the creek.

MR. TRUSKOWSKI: I'm sorry --

BILL NAIL: My well has never been checked that I know of at all, they come there one time and took the top off of it and couldn't get no water out of it, but they didn't take it out of the hydrant. That's the only way you're going to get it out of mine. I would like to see it checked, because I am right on the tail end of where it runs off of your railroad track and out of Cricket Springs both, because I am only a hundred yards off of the creek, and an 80 foot

well, that is all I have.

MR. TRUSKOWSKI: That will be something that we will consider very strongly doing as part of the record of decision as part of the comment period for the proposed plan that we are going to do right after -- probably early April, also, is it goes into a public comment period where you get to read what we want to do and then send in comments that say, "This is what I would like you to do in addition," or "This is what I would like you to do different than this," or "Why are you doing that?"

BILL NAIL: Well, I'm about a quarter up this way, it goes -- it is underground all the way down, past this place here, down underground, all the way down, it is about a quarter before it gets to my house, and it comes out and comes right out on the rocks there. So, my well is very shallow.

MR. TRUSKOWSKI: Eighty feet in this area is actually a fairly deep well.

BILL NAIL: Well, I am sitting in the bed of Cricket, though -- right here is where it is at. I'm right down -- right in the lower part of it.

MR. TRUSKOWSKI: Cricket Creek or Cricket

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Spring?

BILL NAIL: Cricket Creek. Right on Cricket Creek.

MR. TRUSKOWSKI: Okay. Cricket Creek has never shown any contamination. We have never found any contamination in here.

BILL NAIL: Where you think the spring water here, goes? It is going to run into Cricket Creek, and also your railroad track comes right down and comes right out, right there in front of his house.

MR. TRUSKOWSKI: Right. Well, we have never found contamination down there in the sediments or in the water.

BILL NAIL: Is it because you haven't been down there, or you've been down there -- nobody has ever tested my water.

MR. TRUSKOWSKI: The well water?

BILL NAIL: Right. I drink that stuff every day.

MR. TRUSKOWSKI: Probably because of the location. That was the decision that was made earlier, which wells we were going to sample, which ones we weren't going to sample, and we did it based on where the well is to what water unit

it is getting the water out of, and where in proximity to the site.

BILL NAIL: Well, at my age, I don't want to die with cancer. I have still have got fifty years to go.

MR. TRUSKOWSKI: Trust me, I don't want to die of cancer, too.

JOHN PARTON: He brought up a point there about running off the railroad. A few months back, my son and myself began at Cricket and we walked about four miles on the railroad out to Crest tunnel. All along there had been a railroad car there that evidently had broke loose or something, and there was just a stream of oil right in the middle of the ties all the way out. So, if you find something, say, at his place, how are you going to be guaranteed that it didn't come from the railroad? How are you going to be guaranteed that some tanker car didn't come through and it had a break in it and diesel went all over the railroad and washed off the side and down the creek?

MR. TRUSKOWSKI: We went down to the site today, I was showing Gary some of the features of the site today, and we were walking along the

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railroad, and, yes, that is exactly right, there are spots of oil on the rails, and you and I were talking about that a little earlier, too. I, you know, the site in this remedial investigation --

JOHN PARTON: The more miles we walked, every bit of it goes off and down the Cricket to the place where he was talking about.

MR. TRUSKOWSKI: There is one point along one of the channels, on the, I guess, the north side of the road right down here, where this side of the road, down here, when we were walking, me and my contractor were down there looking at it, and, yeah, there was a very oily place. Appreciate you coming.

EDSEL BAKER: Thank you.

MR. TRUSKOWSKI: I hope I helped you out.

EDSEL BAKER: Yeah.

MR. TRUSKOWSKI: There is a seep of oil coming out from there that we had them sample while they were out, and it didn't show up anything that would relate to the site, either, which tells me that it probably came from the railroad.

JOHN PARTON: There is a railroad tie about every two foot or foot apart and it is also

treated with creosote.

MR. TRUSKOWSKI: Creosote, right.

JOHN PARTON: That the water would wash onto it and soak down.

MR. TRUSKOWSKI: That's right. And there is no way we can tell that contamination isn't coming from that.

EDSEL BAKER: But even that (Inaudible), not on Cricket (Inaudible).

MR. TRUSKOWSKI: We did it today, and we saw the sheen on the top --

WILLIE TATE: You could tell it was go between (Inaudible) when the plant was in operation.

MR. TRUSKOWSKI: Can you?

WILLIE TATE: When it was in operation and how far the -- along the road, to the creek.

MR. TRUSKOWSKI: You look at the volume that must have been going down into the -- just into the dirt, the amount of the oil.

WILLIE TATE: I don't know, but it seems like it would dissipate a little bit along the line. It might not ever -- (Inaudible) -- it.

MR. TRUSKOWSKI: That is what the investigation has showed.

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LADY: If you can smell that when it goes through, and it is -- (Inaudible) -- to water it down, and how much of that is on the rocks.

MR. TRUSKOWSKI: That's a very difficult question to answer. Pass the buck again. That's something that really a toxicologist would need to be able to answer. That is one of those questions of what the penta, what the oil that is in the ditch, how toxic it is, and -- (Inaudible). I just don't know, I can't answer that question.

MRS. ADAMS: In the creek -- (Inaudible).

MR. TRUSKOWSKI: Yes, we have. We have tested the sediments.

MRS. ADAMS: How far down?

MR. TRUSKOWSKI: All the way down Cricket Creek, and down -- (Inaudible).

LADY: How long did you do that?

MR. TRUSKOWSKI: How long since we sampled it?

KEN ADAMS: Yes, --

MR. TRUSKOWSKI: It has been since that one high rain that you were talking about earlier, and not recently.

KEN ADAMS: Not recently. Well, you don't

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know how much has come through, how much is stuck to the gravel, and how much has been washed into the yards down there, how much has stuck -- how much has run, water has run off into the ground. Some people, then they are going to -- and gardens, your plants take all of the nutrients out of the ground, well, that is going to be in there, the dioxin and all of that, you are going to eat that, too.

MR. TRUSKOWSKI: All I can tell you is what the remedial investigation showed up. None of the sediments along the creek or along the spring channel that feeds into the creek have shown any contaminant. I realize I have seen it, too, I have seen the oil on -- a sheen of oil on the water sometimes and on the rocks, too, and you can smell it when you are down in that area. But the testing hasn't shown up any penta or any PNAs in that ditch, in any of the sediments.

JUDY BOYD: Did you check the water level with -- (Inaudible)?

MR. TRUSKOWSKI: Not the sediments, the water, tested the water when it was high?

JUDY BOYD: And you tested the sediments when the water level was --

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MR. TRUSKOWSKI: I believe it was in a fairly low stage. You know, you try to grab where you see the most contamination. It doesn't matter where the water actually is because what happens is the oil, when it floats on the top, it will go up to the highest level while it there, then when the water goes back down, it will coat the sides there. So, what we try to do is try to sample along there, to try to find out what it is.

So, by the middle of April, we will have a proposed plan also in the repositories, which will tell you what we are thinking about doing out there, and it will be opened up for public comment in which you will read the plan, and you also will have the RI/FS in front of you, which you can read also. We will take the comments and observe, you know, if your comment is, "I would like to see my well added as part of the monitoring."

BILL NAIL: Yes, I would.

MR. TRUSKOWSKI: We will take a look at that and look at your well and see if we can do it.

BILL NAIL: Are you going to have a reading on it before you decide what --

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MR. TRUSKOWSKI: Right. After the proposed plan comes out, we will have a full scale public meeting, not like this where I'll be up against a target where you can throw things, but we will be up -- but we will give you will another chance to ask more questions. But this will be based on having the remedial investigation and the feasibility studies in front of you, and the proposed plan, so you will have all of the information that I have in front of you in order to ask the questions, and then we will take the comment that -- the comments that you send during that three day period and we will incorporate them into the decision-making process.

Right now, we are hoping to get a final decision on what we are going to do at the Arkwood site out by the end of June, and we will take the proposed plan and take the comments and incorporate them and the record of decision may or may not be the same thing as the proposed plan depending on what comments we have from the public.

MRS. ADAMS: Does that mean actual starting in June to cleanup, or what?

MR. TRUSKOWSKI: After the -- after we sign

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the ROD, we will go into negotiations with Mass Merchandisers to -- for the conditions of the cleanup.

MR. BONDY: (Inaudible.)

MR. TRUSKOWSKI: Yes, I am trying to go over that all.

MRS. ADAMS: How long will it take?

MR. TRUSKOWSKI: We will be looking at the design starting approximately six months after the negotiations, that is typically --

MRS. ADAMS: The design starting.

MR. BONDY: Before you start the design, how long the design will take, I don't know. We've had designs take a couple of years.

RAYFORD DUGGAN: Are we talking about a blueprint or something like that?

MR. BONDY: Exactly.

RAYFORD DUGGAN: We are not talking about the actual work?

MR. BONDY: Exactly.

RAYFORD DUGGAN: Well, what are we talking about long-range?

KATHY DUGGAN: We are talking about -- (Inaudible.)

MR. TRUSKOWSKI: Again, it is hard to tell,

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because it all depends on what the remedy is.

RAYFORD DUGGAN: From what we were told the last time, you are already two years behind at this point here, they said ten years then, so, 20 years can be affected.

EDSEL BAKER: (Inaudible.)

JUDY BOYD: If people decide that they would like the dye tracer, and feel that the diagnostics have really not been complete enough

MR. TRUSKOWSKI: Right.

JUDY BOYD: Do they get their input on that before you develop a proposed plan, or are you going to do your proposed plan and wait and see if people feel strongly about having the dye tracer done on the water flow?

MR. TRUSKOWSKI: I can say yes to both questions. Yes, it might be part of the proposed plan, and yes, it might be included in the ROD based on all of the comments. I don't know yet.

MR. BONDY: Now, if you are aware of something, to help yourself out, you can just -- you can request us to do something, some kind of testing, then you really need to justify why you want us to do it, why it should be done, not just because you want it done, otherwise. We have to

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have a reason, not just a basic request, we really need to have a reason why it should be done. We think it needs to be done because you don't know this, and this could maybe tell you this type of thing; that would really strengthen your letter to us.

JUDY BOYD: Most of us here are real familiar with the case in Green Forest with the sewage problem where eventually they did do a dye tracer, and it carried far, far, far beyond what anybody thought that it would, and it totally changed the a situation as far as what they needed to do in developing a treatment plant in Green Forest, and also, totally changed the claims that people could levy against Tyson's that had health problems and deaths in the family that were attributed to it. Had they not done that, the people that could claim injury or death would not have been able to claim it; they wouldn't have been able to prove that the water flow did carry to their own system. So, in not doing that, you automatically lock out the future potential for people to make claims.

MR. TRUSKOWSKI: The dye tracers can be very powerful tool in Karst; that has been recognized

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in this region especially. I think Arkansas doesn't even allow landfills to put in dye tracer studies. It's a very powerful tool.

JUDY BOYD: Do you think that as much money as we are talking about here, and the kind of extreme risk to people that we are talking about here, and the amount of years that we are talking about that this will affect people in this area, is that such a small thing to do that will have such a big effect in the long run? I don't understand why that wouldn't be done.

MR. BONDY: One of the big things why we are not sure it it is going to tell us anything, we have got to be able to find this dye. When we inject it into the ground water, we have got to be able to to find it. We are not so sure we will be able to find it.

JUDY BOYD: Well, you will never know unless you try.

MR. BONDY: That's right.

MRS. ADAMS: Just knowing that is important. Not finding it. We won't know where contamination comes.

WILLIE TATE: We are going to know about the wells. We'll know it didn't go in our well.

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MR. BONDY: We kind of already know that.

JUDY BOYD: If it shows up in 200 wells spread out over the area, it is definitely something that we need to know now.

MR. BONDY: Yes.

JUDY BOYD: Rather than later when we, you know, can no longer --

MR. TRUSKOWSKI: Well, as part of the remedial investigation, we have had plans for a dye tracer study done. They were in the investigation work plan, the phase two work plan to be done, and we even went so far as to get a plan from M.M.I. for doing one. But after consulting with the geologists and the hydrologists that we have on staff and their and our oversight contractors, the timing is wrong. That is what it really comes down to, is that we don't have the ground water condition, because of the drought, to do one that would really tell us what we need to know. And what we have got is we have got some very flow rates through the aquifers, the conduits where the water is flowing, and so, as a result, we are not seeing the contamination coming out in a lot of places that might, if the water level was still up.

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JUDY BOYD: Well, it seems like from your perspective, then, that that would be the time to do it, because if it is going to show up in people's wells under those conditions, than it would later under optimum conditions.

MR. TRUSKOWSKI: It depends on what you are trying to find out. If you are trying to find out what happens during the low flow --

WILLIE TATE: We are trying to find out where the water flows to.

MR. TRUSKOWSKI: Right. That is exactly right.

JUDY BOYD: And considering the situation, if it would be reasonable even to do it under less than optimum conditions and do it again later under optimum conditions. We are talking about people's lives here, and integrity of the land for hundreds of years. It seems a small thing to do. Even if you have to do five tests, it seems well within reason, when you are talking about spending millions of dollars on a -- (Inaudible).

You know, we have here at the school, there is a book down in the library, and it is on geology, and one of the things that it lists in

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the back of it is the, you know, top toxic sites in the country. And I guess this is, what, among the top 13, of the most -- our book down here. Honestly, our book down here lists it as Number 13 in some degree of something, I'm not sure what, but that is nation-wide.

MR. BONDY: Well, I didn't see that, but you

JUDY BOYD: Honestly, the kids brought it to us and showed it to us, and this is a book that was printed last year. And, you know, even were that not true, it is obviously a serious enough problem that millions are going to be thrown in to remediate the problems, and it just seems like a very small thing to do on the front end to make sure the problems fit to an adequate livable, reasonable level, rather than not doing it now. You know, I don't know any cardiologist that would go in and do open heart surgery on somebody without first doing reasonable tests to make sure he knows what he is going in to do, and what is effective, and it seems that that would be one of the elementary things to do.

MRS. ADAMS: As with test wells like theirs. You know, when you're drinking water every day, why do these people have to wait to have thei

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wells tested? Why do they have to get your permission and prove to you and whoever did this that they deserve to have their wells tested? It's enough that they have doubt about their water that they drink every day. Why do they have to prove that to you? Why can't you just come out and do it automatically? It's elementary. It's basic. Give people security.

BILL NAIL: Well, when you are living right on the tail end of the line, you know, that is where it goes to.

MRS. ADAMS: That's ridiculous. They have to put it in later.

EDSEL BAKER: Like you said, it is going west, and I am living due west from the area, right on the Cricket branch, right on the creek, that is a very shallow well.

MRS. ADAMS: That's absurd.

MR. BONDY: You are suggesting that --

MRS. ADAMS: I'm suggesting that people deserve security for something as basic as drinking water when it is not their fault that it has been contaminated, and the least thing whoever is responsible can do is test the water and make them have security. That's basic, isn't

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it?

BILL NAIL: The county just -- they should have had it done before I bought this place down here right in this county of what was going on here. I didn't even know there was a dump site here. That is kind of foolish the way they do things. It looks like they kind of blind man, then get his money, and then sit back and say, "Ha, ha, ha."

WILLIE TATE: Well, that is one of the problems of several people that I know of. They want to sell, and the very first thing, somebody will tell them that the well may be contaminated or something, and they won't even talk with them. It has been that way for four or five years, and I know a couple of houses down there by me that the people come and looked, but they just looked and then when they found out the wells were contaminated, they didn't want to mess with them. When you said it was going to be four or five more years, those people may just not need to sell, then.

MR. TRUSKOWSKI: Again, the wells, you know, haven't shown any contamination.

JUDY BOYD: You tested 12 wells, and you

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don't even know for sure the flow of the water.

BILL NAIL: (Inaudible) -- without even being tested, and it is right on the drain off of wherever things are really going, that is what really burns me up.

JUDY BOYD: And you're talking that you are going to develop a proposed plan without ever doing a dye tracer to see where the water is going and where you need to test it.

MR. TRUSKOWSKI: The dye tracer study may be a part of the design. Again, we don't know, we don't know exactly how we are going to approach the site as yet. After the remedial investigation and feasibility study are done, that is when we will come up with our proposed plan, and there are some very good points that are being made, and I think I would like to see them as part of the public comments that come in after we do it.

BILL NAIL: It ain't going to do any good --

MRS. ADAMS: We just have to keep making them. I mean, nothing happens. We talk and we have another meeting, and then --

MR. BONDY: So, we would have to do that --

BILL NAIL: If I knew what was going on here

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in Boone County and where this dump site and stuff, I wouldn't have ever sunk $50,000.00 into that place, I guarantee you that. It don't make sense.

LADY: You said M.M.I. has been very cooperative. They are one of the primary people who employ people here in this county. I mean, how do they feel about cleaning it up, do they feel an obligation to, or are they, since they employ so many people, going to do politically as they please.

MR. TRUSKOWSKI: No, I think that they do feel an obligation. I think that they realize that they are potentially responsible for it, so they are trying to be very -- that is the feel I personally get from that. Now, I can't -- that is not an official agency stance or anything like that, that's just --

LADY: Well, you are the one working with them, you are the one -- (Inaudible).

MR. TRUSKOWSKI: Yes, I feel like they are trying to do well.

KEN ADAMS: How far east did you go test the water, the ground water?

MR. TRUSKOWSKI: The wells?

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KEN ADAMS: No, just the ground water.

MR. TRUSKOWSKI: As far as --

KEN ADAMS: On the other side of the plant.

MR. TRUSKOWSKI: Sinking the monitoring wells?

KEN ADAMS: Doing anything.

MR. TRUSKOWSKI: I would have to have a map. I believe we were sampling wells even out in this area.

KEN ADAMS: On the far side, down on this side?

MR. TRUSKOWSKI: Off of this side, back down here?

KEN ADAMS: Which would be Walnut Creek.

MR. TRUSKOWSKI: Right. We didn't do any well sampling out there, because --

KEN ADAMS: What about ground survey?

MR. TRUSKOWSKI: Surface survey?

KEN ADAMS: Yes.

MR. TRUSKOWSKI: No, we haven't.

MR. BONDY: We sampled the creek.

MR. TRUSKOWSKI: We sampled the creek.

KEN ADAMS: You sampled the creek.

MR. TRUSKOWSKI: Right. To see if anything was coming out, we sampled the spring coming out

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of the railroad tunnel, water coming out of the railroad tunnel, nothing there, and nothing in the sediments, nothing has shown up on that side.

KEN ADAMS: There are not that many houses along Walnut Creek, that is the creek that runs back the other way from the tunnel.

MR. TRUSKOWSKI: It comes down in here.

KEN ADAMS: In other words, there are not houses built right along the creek right through here, they are approximately out that way.

MR. TRUSKOWSKI: That is all field, that's all grazing and stuff like that.

KEN ADAMS: I know of one spot down there that has diesel, oil, oil cans, laying off to the side of the railroad tracks, and that is just sitting down there.

EDSEL BAKER: Right off the railroad.

KEN ADAMS: Off the railroad. You can see -- I have been through the tunnel, you can see the spring comes out with diesel in it, running down the side of the bluffs, and run off into a hollow. Usually it is right there where the railroad track meets the hollow ditch.

MR. TRUSKOWSKI: I've walked through there, too. I used to work in mines, and we had the

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same thing in there, and I never realized what exactly that was that was coming off the railroad tracks.

KEN ADAMS: I have seen that, I have been here many years, I was raised around here, I have walked through there hunting. I'm 26 now, and that's been ten, 11, 12 years ago. It has been sitting there all this time, the houses, or the water goes through there, there is a few on that hillside, and there's caves back in there, and nobody knows where the water goes through, no geological survey of the water flow. (Inaudible) sediment, caves in there --

MR. TRUSKOWSKI: As a part of the investigation, we have never found anything on that site, that is all I can -- you know, geologists that we have working on the site have indicated the water is going the other way from the site. I don't know how much more -- how much more certainty we could have about the water not going down in that direction.

JUDY BOYD: Well, wouldn't a dye tracer tell you?

MR. TRUSKOWSKI: Yes, it might. It might.

EDSEL BAKER: If properly done?

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MR. TRUSKOWSKI: Be done in the right conditions, also. You know, one of our ideas is that where the water that was contaminating the railroad springs was coming from, was when this area where the treatment yard, the storage yard was operational, what was happening is that the PCP was running out that way and then getting into the water there and coming back into the railroad tunnel going up that way. But once it stopped operating, nothing ever turned up again.

KEN ADAMS: What about the study, that they -- (Inaudible) -- the tunnel?

MR. TRUSKOWSKI: The only -- this one right here, the sink hole, they are talking about?

KEN ADAMS: Right there.

WILLIE TATE: That sink hole that you are talking about, it don't go towards the tunnel, it goes down and comes out that spring.

MR. TRUSKOWSKI: Yes, we think that it is one of the areas that may be charged to. I'm not familiar with the tunnel that you are talking about.

KEN ADAMS: Right next to Arkwood, right next to the plant.

MR. TRUSKOWSKI: The railroad tunnel?

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KEN ADAMS: Yes.

WILLIE TATE: The sink hole, the sink hole drains into those tunnels.

MR. TRUSKOWSKI: You are not talking about the one actually up on the site, the one that they covered over with concrete?

KEN ADAMS: No.

MR. TRUSKOWSKI: You are talking about the one down along the side of the --

KEN ADAMS: Down on the side.

MR. TRUSKOWSKI: I'm not familiar with that at all. I'm not sure where you are talking about.

MR. BONDY: You guys are missing each other.

MR. TRUSKOWSKI: Yes, I think we are missing each other.

KEN ADAMS: A bunch of oil and stuff right by the railroad tunnel. I had heard that there was an old mine shaft or an old cave next to the tunnel which they found barrels of dioxin and creosote down in there.

MR. TRUSKOWSKI: Okay.

KEN ADAMS: It may have been dozed and caved in.

MR. TRUSKOWSKI: I think I know where you

are thinking of. When I first took over the site from the past RPM, one of the things I always do when I get a site I am being assigned to is go through the files and read everything in the files that I can and make sense out --

KEN ADAMS: This may not have been in the file.

MR. TRUSKOWSKI: And what I found in the files was the report of a cave that they used to dump in, dump the sludge in. That turns out to be this sink hole right here. And what they dumped in there was the sludges, plus some wash water, and then covered it up with concrete and stopped using it when ADPC&E, Arkansas Pollution Control and Environment, found out about the contamination coming out of the railroad spring. And I think that is where -- I think that is what you are heard about, is this right here. It was called a cave at one time that they covered up, a cave that they dumped in, but what it is in actuality is a sink hole that they used on-site as a convenient dumping place. Now, back then there was a hole in the gound right here and you pour your water in and it goes away. Imagine that. But what they had was a place for

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disposing of wastewater, I think that is what you are talking about. That is the only cave that I have seen or heard about, even when I walked down next to the tunnel. That is the only thing that I can think of that you could be talking about.

KATHY DUGGAN: You said there were --

MR. TRUSKOWSKI: Go ahead.

LADY: Why did you decide against off-site incineration of some of that soil?

MR. TRUSKOWSKI: We haven't made a decision yet about off-site incineration, on-site incineration, washing the soils, biological treating of the soils, we haven't made a decision.

LADY: (Inaudible.)

MR. TRUSKOWSKI: Oh, the removal action? Yeah, what happened there was M.M.I. proposed -- as part of an investigation, the obvious -- one of the obvious sources is that railroad ditch area, you know, I said that is where the highest health threat is from, that is right in here, this area right in here. And what it used to be was sludges and wash water was poured down in there. As part of thei investigation, they requested that they could do incineration. Quick

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and dirty, this is the bad source, let's take it up, let's burn it.

As we got closer and closer to the end of the remediation, it became really difficult for M.M.I. to find somebody to take it, and the waiting list on the incinerators is fairly long, also. And so, it is just a case of the logistics of it. It made as much sense to do it as a part of the remediation as anything else. It was a joint decision made. M.M.I. basically pulled back their request and we weren't going to force the issue, because we weren't prepared to do it ourselves.

KATHY DUGGAN?: So, you are saying that that is not out, it is just delayed.

MR. TRUSKOWSKI: Right, right. It is probably going to be just delayed. In fact, most of the remedies that we are looking at include the incineration of that material there. So, it is a very real possibility that that material is going to be burned, eventually.

KATHY DUGGAN: Any possibility that it would be burned on-site?

MR. TRUSKOWSKI: Very small, very small. I can't say yet; we haven't made the decision what

we are going to do there.

WILLIE TATE: We probably won't know anything until after this final report, then?

MR. TRUSKOWSKI: When the proposed plan comes out, that is when you will get our best idea of what we want to do out there, what E.P.A. wants to do to best remedy the site.

JUDY BOYD: What is required for final approval on that plan? Is that part of the -- (Inaudible) -- in the proposed plan?

MR. BONDY: What are the criteria there?

JUDY BOYD: At any point, can the community say, it doesn't meet our standards, and go back to negotiating?

MR. BONDY: First off, your question was, how do we select the remedy, what goes through making the final decision of okaying the remedy, is that what you asked?

JUDY BOYD: Right. You are about to develop a plan, and then the people in the community will give input. What kind of input, and what kind of power does the community have? Do you do that and then go ahead and do what you planned, or does the community, the people involved, do they approve of the plan?

009014

MR. BONDY: There are a total of nine criteria to be looked at. The first two are called the threshold criteria. And the alternative has to meet those, the protection of public health and meeting all appropriate regulations and laws. Then it has to -- then it goes into a bunch of other things, long-term effect, short-term effect, and on and on. Two of those in there are state, the state government's comments on the plan, and then the community acceptance of the plan, called community acceptance.

JUDY BOYD: What kind of veto does that have?

MR. BONDY: It is not a veto, I will tell you that, if that's what you're asking. Let me tell you --

JUDY BOYD: What kind of effect, on or what -- does the public input has -- is that one and the same --

MR. BONDY: It is a real gray type of thing, involving a lot of judgments, so it is a real difficult thing to answer, what you are asking me. The way it works we is go to the public with a proposal for the public, and if the public

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raises a lot of legitimate concerns, it is very gray. Legitimate concerns as judged by E.P.A., okay?

JUDY BOYD: So, there is no set standard nationally --

MR. BONDY: That's right.

JUDY BOYD: -- of what degree of public concern is enough concern to change the plan?

MR. BONDY: That's correct.

JUDY BOYD: So, it's really left up to E.P.A. to decide whether the degree of public concern shown is enough to generate for more negotiations?

MR. BONDY: Right. Yes, you are saying negotiations, and I am kind of catching on that, I'm not sure what you mean by negotiations.

JUDY BOYD: Well, people in the community have expressed the desire to have some more diagnostic studies made than have already been made.

MR. BONDY: Uh-huh.

JUDY BOYD: My specific question, and you say one of those nine points involve community input, how is it measured, what degree of input can ensure that their desire to have these other

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tests -- (Inaudible).

MR. BONDY: You are kind of mixing two things. One, the remedy of the site, what are we going to do to fix the site; and two, you want other tests to be run.

JUDY BOYD: Yes, and -- (Inaudible).

MR. BONDY: The two do come together eventually, if additional tests would affect the remedy or not. You are kind of asking me two things.

JUDY BOYD: Well, does that mean, then, that these additional diagnostic tests should be made before the plan is drawn up and proposed?

MR. BONDY: What it comes down to is whether we believe we have got enough information to select the remedy. Right now, we believe that we do.

JUDY BOYD: And if the community believes that you don't have enough information, then what do they do to generate a change?

MR. BONDY: Write to us is the best way, write to us.

WILLIE TATE: She is probably trying to find out --

MR. BONDY: One thing I want to bring up is

009017

we are accused of studying forever, and now you want us to study further. What we do is try to gather the amount of information that is necessary to characterize the extent of the contamination and go and do something about it. That is where we draw the line. You are asking us, where do we draw the line, there is a gray area where we draw it. We use our judgment to try to -- okay, we do all of these tests to figure out how much information do we need in order for us to make a decision on what needs to be done, how much contamination is out there, where is it going. And we feel we have got enough information to do that right now.

JUDY BOYD: So, E.P.A.'s judgment is that dye tracers are not necessary to ensure the people where this stuff is going -- (Inaudible). It is not E.P.A.'s decision that that is necessary to have enough information to make their plans, is that what you are saying?

MR. TRUSKOWSKI: You are getting into the area of how confident we are with the information we have.

JUDY BOYD: Right.

MR. TRUSKOWSKI: We are fairly confident,

009018

yes. I think a dye tracer study would give us some more information that would be very helpful, make you feel a lot better, make M.M.I. feel a lot better, make E.P.A. feel a lot better about what we have got out there, and how well we know where things are going. What I am saying is that at this point, the conditions are not right to do dye tracing and get the information out of it that would make anybody feel more comfortable.

JUDY BOYD: Well, we are heading into spring where the water level many be a totally different thing here.

MR. TRUSKOWSKI: Right. I hope so, I sure hope so. And my yard is badly in need of water right now. You know, we have got a couple of good rains. Down in Dallas, it is so dry, that lake levels are starting to drop. It is not a good situation, all in all. See, one of the problems with doing the dye tracing when the water is real low is that in the Karst, when the water goes up, it hits more conduits, more going out, so the best time to do a dye tracing would be just as high as possible. And, unfortunately, we don't have that situation.

JUDY BOYD: Up in Green Forest, you know,

009019

they had to do theirs under less than optimum conditions, too, and it still gave them much more information than they would have had without it.

MR. TRUSKOWSKI: We may be forced to do that, also; we don't know yet. We are hoping to be able to do it at optimum conditions. You know, even if we did one now, we wouldn't get any information back until after the record of decision, unless we put off the record of decision, which nobody really wants to do, either.

JUDY BOYD: Well, now, wait a minute. Nobody in E.P.A. The people in the community may feel differently.

MR. BONDY: Let me answer that. We make the decision on the record, and when we get out and get more information later, we can always change the decision on the record. It has been done, It has been done in the past. So, once the decision is made, that is not it, okay, we get some more information.

WILLIE TATE: I would kind of like to know, myself, say you do some more tests and you say, okay, I don't think for three years, now, it has went down and made a few more, and we got a

009020

little more, and maybe we ought to just forget the whole thing. Can the community have anything to say?

MR. TRUSKOWSKI: Well, part of the -- one of the things that we are really sure of is that as part of the ground water remedy, it would be monitored for about 30 years. So, you know, we wouldn't do it for a couple of years and say, "Well, nothing is showing up, nothing is happening, so, let's just call the whole thing off." We will keep doing that for about 30 years. And during any time in there, if there is something showing up or we get a way that we can find out where the water is going, say, technology becomes available to show that, hey, we can get this problem taken care of just like that, it is always possible to go back in and say, we are going to do it. That is always a possibility, it is only on the back burner.

WILLIE TATE: It is a possibility, you have the additional, a few of those of hot spots -- (Inaudible).

MR. TRUSKOWSKI: We think that may happen, also. But you know, like you said, you never know until you actually get out there and do it.

009021

JUDY BOYD: I just think one of the main concerns is when you continue to monitor the ground water for years, if you do it after the way you have been doing it, 12 wells in a very limited area, then you are not covering the concerns that you test over a wider area, or at least do some diagnostic tests to know that you don't need to cover a larger area. There has not been anything to prove that you shouldn't expand your tests to a larger area, is that correct?

MR. BONDY: Well, we have never found it off-site, that is why we say --

JUDY BOYD: You never found it off-site, you said, how many tested areas.

MR. BONDY: You asked for diagnostic, and that is it.

JUDY BOYD: Well, I would like to know --

MR. TRUSKOWSKI: Well, it looks like -- is the hearing over? I would like to talk to you some more.

VIDEO OPERATOR: Well, the meeting ended by people just getting up and walking away.

(WHEREUPON, the above-entitled public hearing was concluded.)

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009022

C E R T I F I C A T E

STATE OF ARKANSAS)
) ss.:
COUNTY OF PULASKI)

I, DEBBYE L. PETRE, Certified Court Reporter and notary public in and for the County of Pulaski, State of Arkansas, duly commissioned and acting, do hereby certify that the above-entitled proceedings were transcribed by me in Stenotype from video-tape at the request of Mass Merchandisers, Inc., and with such corrections as people familiar with the proceedings have indicated, and the same truly and correctly reflects the proceedings had to the best of my ability.

WHEREFORE, I have subscribed my signature and affixed my notarial seal as such notary public at the City of Little Rock, County of Pulaski, State of Arkansas, this the 13th day of September, 1990.

Debbye L. Petre.

DEBBYE L. PETRE, CCR
NOTARY PUBLIC IN AND FOR
PULASKI COUNTY, ARKANSAS

My Commission Expires:
August 4, 2000.

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009023